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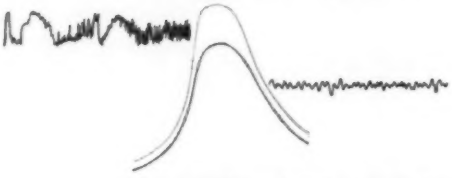

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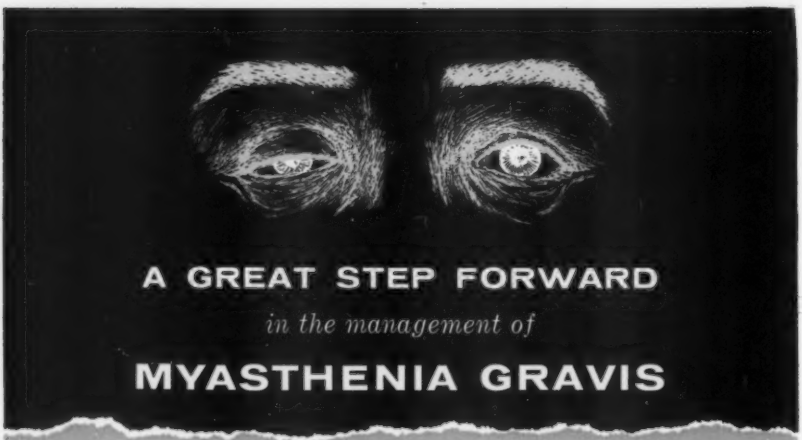

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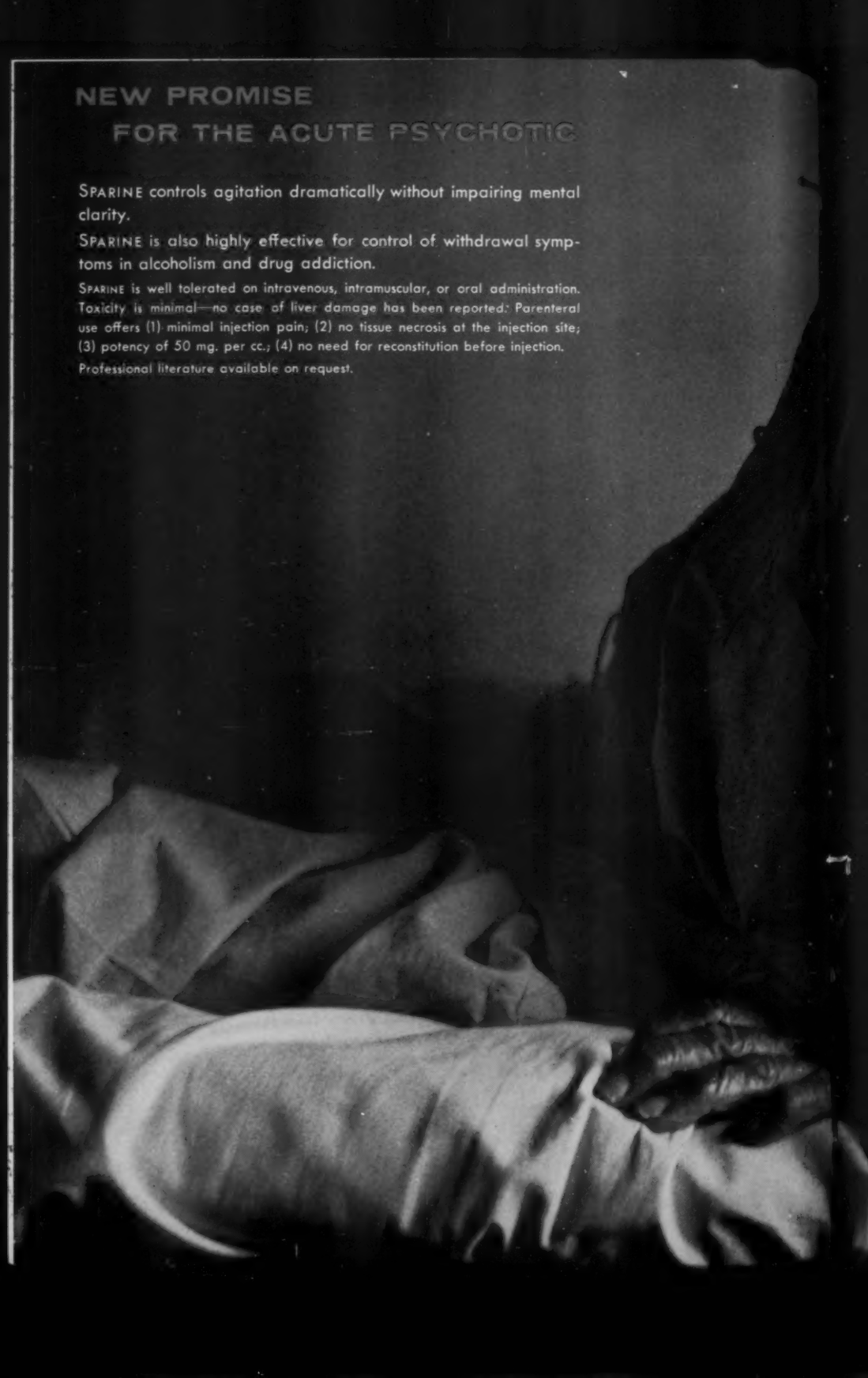
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Volume 7 Neurology *Number 1*

"Psychomotor" or "Temporal Lobe" Epilepsy

A Review of the Development of Our Present Concepts

Russell N. DeJong, M.D.

THE TERM "psychomotor epilepsy" was introduced by Gibbs and Lennox in 1937.[1] In discussing the electroencephalographic features of epilepsy, they referred to "psychic variants" and "psychic equivalents," but also used the term "psychomotor seizures," and in one place the expression "psychomotor attacks" was followed by the parenthetical term "psychic variants." They did not at that time appreciate the focal nature of the process in these seizures, and described what they felt was a typical slow, flat-topped or plateau-topped wave pattern with superimposed normal rhythm still present.[2] Lennox has stated that the authors "felt that the word psychic did not take account of the motor activities that many patients displayed and adopted the word 'psychomotor'."[3] Gibbs has said recently, "We did not wish our confreres to think we were describing something new. We were talking about a clinical type of attack which was common and had long been recognized. However, the terms which we found in common use (ictal automatism, psychic equivalent, fugue) were not easy to restrict and were somewhat misleading. We decided in 1937 that the word *psychomotor* was satisfactorily descriptive."[4]

It was later pointed out that the monopolar method of recording then in use, with its auricular or biauricular reference, extends the discharge produced in the temporal region to the entire scalp and therefore transforms a temporal focus of rhythmic waves into a generalized discharge of flat-topped waves. In 1948 Gibbs and Fuster, by changing their method of electroencephalographic recording, were able to demonstrate that in nearly all cases of

From the department of neurology, University Hospital, Ann Arbor, Michigan.
Presented, with minor alterations, as the presidential address at the annual meeting of the American League Against Epilepsy, New York City, December 8, 1955.

psychomotor epilepsy there was a focus of spike potentials localized in the anterior temporal region of one or both sides of the scalp.[5] Penfield and his coworkers, however, even prior to 1937, had noted that in some patients stimulation of the temporal lobe cortex at operation would give rise to illusions of perception, hallucinations, "memories," and even automatisms.[6] Such responses were only obtained in patients suffering from epilepsy characterized by such manifestations. In 1941 Jasper and Kershman, in Penfield's laboratory, observed sharp waves in the temporal regions in cases of psychomotor epilepsy, and they attributed these as well as the ictal symptoms to a neuronal discharge located in the temporal lobe and the underlying structures, or even in deeper structures situated in the vicinity of the median line.[7] More recently Hill, basing his conclusions on a series of 5,000 consecutive electroencephalograms taken at the National Hospital, Queen Square, and the Maudsley Hospital in London, and on studies made jointly with Drs. Denis Williams, W. A. Cobb, and D. A. Pond, re-emphasized the relationship of the functions of the temporal lobe to such seizures, but stated that this relationship is a complex one.[8]

There has long been controversy, however, over the classification of this type of epilepsy on either a clinical or electroencephalographic basis. There may be such variation in the pattern of the attacks that many clinicians feel that all the manifestations should not be classed together. As recently as 1952, McNaughton stated, "There is still insufficient basis for a sound classification of psychomotor seizures. Even the term 'psychomotor' seems hardly adequate to describe the varied forms of abnormal seizures observed in these patients."[9] Hill has said, "Psychomotor epilepsy is not a unitary clinical syndrome. Different authors use the term to include different phenomena."[8] There is an increasing tendency to use the term "temporal lobe epilepsy" as a synonym for the psychomotor type, or to refer to the seizures characterized by hallucinations, memory disturbances, and automatisms as "temporal lobe" in preference to "psychomotor." This has been done even by those who have demonstrated both clinically and experimentally that structures other than the temporal lobe itself may be involved in the process, namely the uncus, hippocampus and hippocampal gyrus, the amygdaloid complex, the posterior orbital surface of the frontal lobe, and the insula. Lennox, for instance, who was one of the originators of the term psychomotor, has said, "At some future time, 'temporal epilepsy' might supplant the present confused terminology."[3] Penfield and Jasper, though both state that the discharges may come from areas other than the temporal lobe, apparently favor this latter anatomic designation.[6] Penfield has even suggested that the term "psychoparetic" might be better than psychomotor, because there is actually a paresis of neural integration,[10] and states, "We would include Jackson's uncinate fits, his dreamy states, and the automatisms, in the somewhat more embracing group of temporal lobe seizures,"[11] even though he has shown that the epileptogenic focus responsible for automatisms is sometimes found in the parietal or frontal lobes or in the Sylvian area, rather than in the temporal lobe itself.[12] Jasper states, "Psychomotor epilepsy is not a specific form of epilepsy but merely one form of cortical seizure arising within the temporal

lobe."[13] Sir Charles Symonds, in an exhaustive correlation of the clinical, electrophysiologic, anatomic, and pathologic aspects of the syndrome, prefers the term temporal lobe epilepsy,[14] as does Falconer.[15] Gibbs, on the other hand, states that, while from studies on the surface of the head the discharge appears to be in the anterior temporal region, recordings at operations show that this area is part of a system consisting of the mesial and inferior surfaces of the temporal lobe, and that these are in close functional relationship with the posterior orbital surface of the frontal lobe.[4] Gastaut, in discussing the subject, says, "It is high time to regard psychomotor epilepsy as an individual entity, of which there are possibly as many varieties as there are forms of cortical epilepsy at the convexity. It is not impossible that in the future a differentiation, based on anatomic and clinical findings, will be made between a masticatory epilepsy and an affective epilepsy or between an amygdaline and hypothalamic epilepsy, in the same way as a somatomotor and visual epilepsy or a prerolandic and occipital epilepsy are differentiated today."[16] Furthermore, he classifies psychomotor epilepsy into temporal, hippocampal, and diencephalic varieties.

The clinical manifestations of that complex of symptoms which we now consider to be those of psychomotor or temporal lobe epilepsy have long been known. In the Bible we read that Balaam "saw the vision, falling into a trance, but with his eyes open."[17] Hippocrates, in "On the Sacred Disease" (about 400 B.C.), described what may be interpreted as automatisms.[3] Aretæus the Cappadocian (second to third century A.D.) described visual, auditory, and olfactory hallucinations "when the term of the attack comes nearer."[18] John of Gaddesden, in the *Rosa Anglica* written about 1314, gives a description of an automatism accompanied by movements of the mouth.[4] Benivienius in 1507 described a patient who had periods of automatic behavior and later developed convulsions.[19]

Hughlings Jackson, whose name we associate with a particular type of focal motor convulsion, was probably one of the first to describe psychomotor seizures in detail, and, furthermore, to localize the pathologic nidus to the temporal lobe of the brain. In 1873 he described olfactory and visual hallucinations in epilepsy,[20] and in 1875 in a discussion entitled, "On Temporary Mental Disorders after Epileptic Paroxysms,"[21] he reported many cases of so-called temporary insanity in epilepsy, either occurring after epileptic paroxysms or "replacing" a fit. He described automatic movements carried out unconsciously and for which the patient had amnesia, and used the term "mental automatism." In many instances he discussed sensory auras of various types, and mentioned one boy whose attacks began with a subjective sensation of smell, following which he would "go out of his mind;" this patient frequently had rage states during his spells and would carry out various automatic movements such as rising and unbuttoning his waistcoat and trousers. In the following year Jackson referred to the "intellectual warnings of epileptic seizures" and first used the term "dreamy state;" he recorded quotations from patients such as "Old scenes revert;" "A panorama of something familiar and yet strange;" "If I were walking alone and had a fit, I should think 'Oh, I saw that before'." He stated that vertigo, gastric sensations,

and transient pallor of the face could accompany such intellectual auras.[22] In his Harveian Lectures on the diagnosis of epilepsy, he described both the "dreamy state" which could occur without subsequent motor or psychic phenomena and the automatisms preceded by various auras, such as epigastric sensations, auditory hallucinations (both formed and unformed), visual phenomena, or gustatory and olfactory hallucinations which might be accompanied by tasting, masticatory, and spitting movements.[23] He stated herein, "I think it will be found that in many, I dare say in most cases, the voluminous mental ('dreamy') state occurs in patients who have at the onset of their seizures some 'digestive' sensation – smell, epigastric sensation, taste, or, in cases where there are movements implying excitation of centres for some such sensations, such movements as those of mastication."

In 1886 and more in detail in 1888, Jackson described what he considered was the first case of epilepsy with an intellectual aura that seemed to be associated with organic brain disease, out of some 50 patients with symptoms of this type that he had seen.[24,25] He described in detail the various disturbances of thought, perception, feeling, and behavior in this "particular variety of epilepsy," and stated that the symptoms most frequently associated with the dreamy state were crude sensations of smell or taste; elaborate hallucinations of sight or occasionally of hearing; epigastric sensations; movements of the lips and jaws resembling those of chewing, tasting, or spitting; aimless movements of the limbs which were coordinated and not convulsive; and, finally, "exceedingly complex and very purposive-seeming actions during continuing unconsciousness." One patient developed papilledema, attacks of left-sided tremor, and left hemiplegia, and died; while there was no autopsy, Jackson hypothesized that the patient died from hemorrhage into a vascular tumor of the right temporosphenoidal lobe. He stated, "He who neglects the 'dreamy state' because it is indefinite and 'merely curious,' and such symptoms as chewing, etc., movements, and apparent alterations in the size and distance of external objects, because they seem trifling things, may not ever surmise that his patient has the serious disease epilepsy in a rudimentary form until a severe fit comes to tell him so." One year later, with Dr. C. E. Beevor, he confirmed the diagnosis in a similar case.[26] This patient had formed visual hallucinations, olfactory sensations, feelings of being shut up in a box, and involuntary urination; she later developed memory loss, paralysis of the left arm and leg, stupor, and papilledema. She was found to have a tumor "at the extreme anterior end of the right temporo-sphenoidal lobe," and "especially the part which is in the front of the uncus of the hippocampal or uncinate convolution, and which contains the structure known as the nucleus amygdalae" (figure 1).

These papers were followed by many others by Jackson and other clinicians giving both clinical descriptions and autopsy confirmation in cases of this type. Of special interest is a physician who, in 1870, writing under the pseudonym Quaerens, described some of his own symptoms.[27] For some years he had only brief "recollections" and "reminiscences," but later developed major seizures as well as automatisms which might be preceded or accompanied by smacking movements of the tongue and

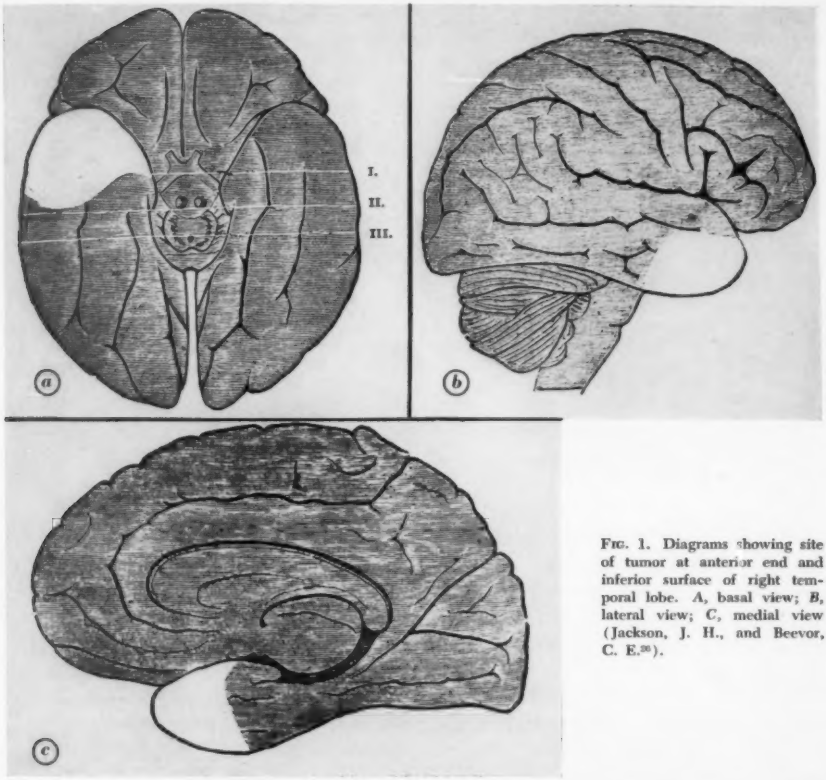

FIG. 1. Diagrams showing site of tumor at anterior end and inferior surface of right temporal lobe. *A*, basal view; *B*, lateral view; *C*, medial view (Jackson, J. H., and Beevor, C. E.[26]).

jaw; there were no actual hallucinatory phenomena.[25] The patient died in 1894 at the age of 49, 23 years after the onset of his symptoms, from an overdose of chloral. At autopsy a small area of softening was found in the left uncinate gyrus just in front of the recurved tip of the uncus (figure 2).[28] This resembled lesions found long after thrombosis or embolism, but no occluded vessels were found.

In 1899 Jackson suggested that attacks with these various manifestations be called the "uncinate group of fits" on the hypothesis that the "discharge lesions are made of some cells, not of uncinate group alone, but of some cells of different parts of a region of which this gyrus is a part – the uncinate region."[29] However, the association of the olfactory hallucinations with auditory and visual phenomena, dreamy states, automatisms, and so on was either disregarded or failed to impress clinicians generally. Gowers, in the second edition of his famous monograph on epilepsy, mentions only that the dreamy state is sometimes associated with special sense aura and refers inaccurately to Jackson's concept thus, "Dr. Jackson proposes for

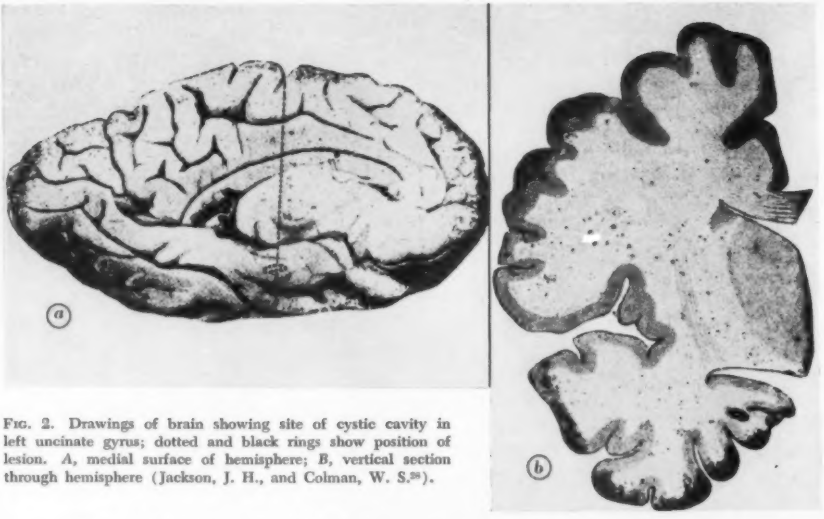

FIG. 2. Drawings of brain showing site of cystic cavity in left uncinate gyrus; dotted and black rings show position of lesion. *A*, medial surface of hemisphere; *B*, vertical section through hemisphere (Jackson, J. H., and Colman, W. S.[28]).

the attacks, with symptoms of the perception of flavour, the name 'uncinate convulsions,' but we are only justified in referring the process to this region when there is evidence of a definite olfactory sensation."[30]

While it is interesting and instructive to review Jackson's development of the concept of "uncinate epilepsy," we all know that the more thoroughly the literature of any subject is studied, the more difficult it becomes to ascribe priority. And credit must be given where it is due. In 1874, 12 years before Jackson suggested the presence of tumor of the temporosphenoidal lobe as a cause for attacks of this type and 15 years before his first confirmation, Sanders, in Germany, reported the case of a patient with olfactory hallucinations, stereotyped movements, mental symptoms, and convulsions, in whom there was a tumor destroying the left olfactory tract.[31] In 1882, 16 years prior to Jackson's publication of the autopsy findings in the physician with an area of softening in the uncinate gyrus, Hamilton, in this country, reported the case of a 40 year old woman who since the age of ten had had attacks preceded by a disagreeable odor and accompanied by choking sensations. At autopsy, in the lower part of the right temporosphenoidal lobe there was "decided shrinkage of tissue, with depression and adhesion of the pia, the induration involving the uncinate gyrus and parts of the adjacent convolutions" (figure 3).[32] In 1887, two years prior to Jackson's first tumor confirmation, Anderson presented the case of a 23 year old man whose symptoms consisted of gustatory hallucinations, auditory phenomena, déjà vu, and brief automatisms; he developed bitemporal hemianopia, optic atrophy, and mental deterioration, and was found to have a large cystic tumor around the area of the pituitary fossa with pressure on and a deformity of the left temporosphenoidal lobe (figure 4).[33] However, backed

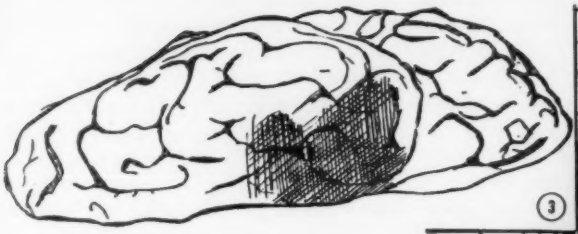

FIG. 3. Sketch of inferior surface of right hemisphere showing area of atrophy in temporal lobe (Hamilton, A. McL.[32]).

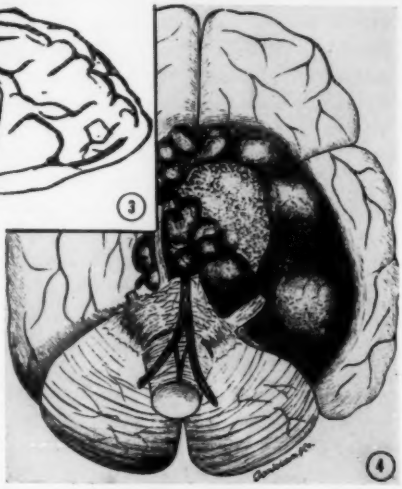

FIG. 4. Drawing of base of brain showing cystic tumor extending into left temporal lobe (Anderson, J.[33]).

by the authority of Gowers, the view prevailed that the single criterion for uncinate attacks was an olfactory aura.[15] As a consequence, the association of seizures characterized by illusions, hallucinations, déjà vu, movements of the lips and jaws, automatisms, and amnesia with abnormalities of the temporal lobe, especially its medial aspect in the region of the uncinate gyrus and amygdaloid nucleus, was more or less disregarded for many years. In 1929 Penfield, discussing Spielmeyer's pathologic observations, which will be mentioned later, stated that the uncinate gyrus (and the cerebellum) were "the two areas which are perhaps farthest removed from the attention of those interested in the epileptic mechanism."[34] However, the electroencephalographic studies of Gibbs and Lennox,[1,2] the clinical observations of Penfield following stimulation technics,[6] and the concomitant electroencephalographic and neurophysiologic studies by his associate, Dr. Jasper,[7] have brought to attention the accuracy of the early observations of Jackson.

The clinical aspects of epilepsy of this type will not be discussed further, as they are well known and have been the subject of increasing interest during recent years. A word or two might be said, however, regarding the incidence of seizures of this type. They were once looked upon as being extremely rare, and Jasper and Kerchman in 1941 reported that only 11 out of 494 epileptic patients studied had "pure psychomotor attacks," an incidence of 9.5 per cent.[7] Lennox, ten years later, in a study of 1900 patients, found that 6 per cent had psychomotor seizures alone, 13 per cent psychomotor and grand mal, 0.7 per cent psychomotor and petit mal, 1 per cent all three types, and 20.7 per cent of the patients had a history of psychomotor attacks with or without some other seizure pattern.[3] In this same group, 80.9 per cent had grand mal attacks with or without other types, and 31.7 per cent had petit mal seizures with or without other types. In patients aged 40 and over, 34.1 per cent had psychomotor seizures with or without some other seizure pattern. Every clinician will agree

that, since we have been more aware of "temporal lobe" manifestations in epilepsy, we are making the diagnosis with increasing frequency. It is a common experience that patients who have been diagnosed as simply "grand mal" or simply "petit mal," on obtaining a more detailed history of all of the details of the attack, reveal "temporal lobe" manifestations even though the entire seizure pattern in many of these patients is not limited to the psychomotor variety.[35,36] Various statistics indicate that anywhere from 30 to 80 per cent of epileptic patients have "temporal lobe" manifestations, even though their attacks may not in all cases be of the "pure psychomotor type."[3,16,37] Furthermore, in the majority of these cases, the electroencephalographic picture shows evidence of abnormality in the temporal lobe area or contiguous structures.[5,38]

The clinical symptoms that make up the various aspects of psychomotor or temporal lobe epilepsy are complex and various. Few patients show all of the manifestations, but an individual patient often shows several of them, and the more diligently we inquire into the history and clinical features shown by any patient, the more numerous are the manifestations we are apt to find in that patient.[15] It is true that many of these have in the past been considered evidences of "idiopathic" epilepsy, but we know that epilepsy is not a disease in itself, but rather a symptom of some underlying cerebral disorder. As our knowledge of it broadens, the percentage of cases formerly called "idiopathic" becomes smaller, whereas those known as "symptomatic" become more numerous. This is especially true in the case of seizures arising in or near the temporal lobes. Studies have shown that epilepsy of the type under discussion can be caused by a wide range of lesions, some of which, such as tumors, cysts, abscesses, scars, and vascular malformations, are macroscopically visible, but others, such as focal atrophy or gliosis, may be detectable only microscopically. The form that an epilepsy takes depends not so much on the pathologic features of the lesion as upon its site and upon the violence of the neuronal discharges in the neighborhood of the lesion and the extent of their spread to the rest of the brain.[15]

In the same manner that current electrophysiologic studies and clinical observations have clarified former points of view and have brought about a renaissance in our clinical localization of so-called psychomotor epilepsy, recent neuropathologic studies have renewed our interest in previous concepts which for many years have been more or less disregarded. In 1825 Bouchet and Cazauvieilh reported sclerosis or softening in the hippocampus of epileptic as well as nonepileptic psychopathic patients.[39] Sommer in 1880 noted that the microscopic changes in these patients were restricted to the band of pyramidal cells in the hippocampus, which has since been known as "Sommer's sector."[40] Bratz in 1899 confirmed Sommer's findings in 50 per cent of patients with "idiopathic" epilepsy, but found the same changes in other diseases associated with convulsive disorders;[41] apparently this was not entirely the result of seizures, as similar changes were found in cases of short duration and in petit mal. Spielmeyer found such focal lesions in the Ammon's horn (and occasionally in the cerebellum) in 80 per cent of 126 epileptics.[42] He described them as small, circumscribed areas of

sclerosis; microscopically there was ischemic destruction, with cell degeneration and thick glial proliferation; Nissl stain showed the change as a loss of ganglion cells in definite areas. He concluded, "One is dealing with the end state. The histological analysis of such sclerotic atrophy cannot give any explanation of the process which produced the atrophy." He did find the first stages of this process in cases of "genuine epilepsy," as well as in examples of symptomatic epilepsy secondary to trauma, intoxiations, eclampsia, and diffuse cerebral processes. He felt that the disturbance was on a circulatory basis and not dependent upon the etiologic process of the epilepsy, as it occurred in different varieties of symptomatic epilepsy. He stated that pathologic anatomy could not give support to the view that there is a "genuine epilepsy." Gowers, in the first edition of his "Epilepsy and Other Chronic Convulsive Disorders" (1881), tried to evaluate these pathologic changes and said, "It is doubtful whether any greater significance is to be ascribed to the induration of the cornu Ammonis (pes hippocampi), to which so much weight has been attached by Meynert. Although the occasional occurrence of this change has been corroborated by several observers, it has been found in only a few of the cases examined, and we are still ignorant whether it is, in some of these, a primary lesion, or is in all a secondary change. The association, by Sommer, of the commencement of attacks by a sensory aura, with this change in a region near where we must look according to Ferrier, for important sensory centres, is, as yet, suggestive only."[43] Lennox and Cobb in 1928 made the statement, "These changes are not expressed in the clinical symptomatology. They give no etiological point of departure. They are only incidental findings which give insight into the sequence of events of the encephalic pathological process."[44]

With recent interest in the surgical therapy of psychomotor epilepsy, attention has again been directed toward pathologic changes in the temporal lobes. Penfield and Flanigin in 1950 reported on 60 cases that had been operated on over a ten-year period.[45] They found atrophic lesions in 49, neoplasm in eight, and no objective change in the three remaining cases. Green and associates in 1951 presented the results of surgery in 23 cases.[46] In six cases there were gross pathologic alterations (meningocortical cicatrix and atrophy); in eight additional cases they found microscopic changes consisting of ischemic ganglion cells and hyperplasia of the pia-arachnoid; and in nine cases no abnormalities were found. Bailey and Gibbs found pathologic alterations in less than half of their 25 cases,[37] but Falconer and co-workers found either gross or microscopic changes in 30 of 31 patients.[47] Meyer and associates, in studying the biopsy findings of 18 cases of temporal lobe epilepsy treated by temporal lobectomy at the Guy's-Maudsley Neurosurgery Research Unit between May 1951 and July 1953, classified their patients into two groups.[48] In the first group of four patients having discrete macroscopic lesions, there were three small tumors and one case of gross posttraumatic scarring. In the second group of 14 cases with noncircumscribed microscopic lesions, the changes varied considerably in quantity and degree. Ammon's horn sclerosis was found in seven cases, with onset before the age of ten in all. In many cases laminary

atrophy, particularly of the third cortical layer, was found and interpreted as a consequence of anoxia. Scharenberg has long been interested in this problem and, by the silver carbonate technic (Hortega), has shown definite cellular alterations of the temporal areas in biopsy specimens of all patients he has studied—not only those operated upon in our own hospital, but also those of Dr. Bailey's series.[49] All specimens were grossly normal, but detailed studies showed necrosis and intense gliosis of the cortex, as well as foci of necrosis and ameboid degeneration of the astroglia in the white matter. Crowe, on the other hand, believes that such changes may be found in patients other than epileptics, and expresses the belief that they are dependent on subjective methods of assessment and may be normal histologic variations.[50] The major pathologic evidence, however, points to definite alterations in structure in the temporal lobe and contiguous areas.

In 1953 Sano and Malamud reopened the investigation of changes in the Ammon's horn region.[51] They examined material from 50 epileptic patients, mainly residents of a state home and epileptic colony. They found pathologic alterations in this area in 29 patients, or 58 per cent. In nine of them there was little significant change other than sclerosis of Ammon's horn. In nine patients there were bilateral changes in the temporal and frontal lobes in addition to the sclerosis in Ammon's horn; the lesions in most of these cases were secondary to trauma sustained during the convulsions, but in some cases were due to trauma prior to the onset of seizures. In 11 patients there were congenitally malformed brains with history of severe mental deficiency. In reviewing the clinical manifestations of these patients, they found that a high percentage had had ictal psychic phenomena with or without rage states or amnesia, postictal confusion, persistent psychotic manifestations, or emotional changes between attacks. The incidence of such phenomena was much greater in patients with Ammon's horn sclerosis than in those without. Furthermore, some of the patients on whom electroencephalographic records could be obtained had shown evidence of temporal lobe discharges. They stated, "In our series it was clear that the patients showing ictal psychic phenomena had had lesions either in the hippocampal formation or in the temporal cortex, or in both, although the reverse was not always true."

The etiology of these alterations and their selectivity for the temporal lobes next aroused attention. In 1953 Earle, Baldwin, and Penfield studied histologic changes in 157 patients with seizures originating in the temporal lobes.[52] In 57, or 37 per cent, there were evidences of postnatal pathology, usually in the form of tumors or sequelae of trauma or infection. In 63 per cent, however, they found the temporal gyri shrunken, yellow, and avascular, and histologically there were increased astrocytes, together with destruction of ganglion cells. This "sclerosis" ranged from involvement of a single gyrus to affection of the entire temporal lobe, but the mesial and inferior portions of the lobe, the uncus, hippocampal gyrus, and first temporal gyrus were the areas most frequently involved. They expressed the belief that these changes, which they term "incisural sclerosis," resulted from herniation of the hippocampus and temporal lobe into the tentorial opening secondary to moulding of the head during

parturition, and that the causative factor was either direct compression of the structures or local anoxia due to compression of the anterior choroidal and middle and posterior cerebral arteries against the tentorial edge. These authors stated further that the lesions which result from such birth injuries "ripen into epileptogenic foci so slowly that temporal lobe seizures may make their appearance many years after birth."

The above concept is an intriguing one, but, of course, it has not been accepted by all workers. Meyer and his associates, for instance, believe that birth mechanisms do not constitute the major etiologic factors, and that events such as infections later in infancy, childhood, and adolescence play as great a part.[48] Gastaut, while he accepts Penfield's views regarding the presence, mechanism, and significance of incisural sclerosis, believes that closed cranial injuries, purulent infections from the mastoid regions and sinuses, viral encephalitis in childhood, and localized vascular lesions are also responsible for a large percentage of cases.[16] These various recent pathologic studies, however, point out a very definite relationship between lesions of the temporal lobe and contiguous structures and the presence of psychomotor seizures, and, further, contradict the once-held view that sclerosis of the cornu Ammonis, when it occurs in epileptics, is always secondary to severe major epileptic seizures.

This discussion would not be complete without reference to the anatomic basis for and experimental evidence of seizures of a "psychomotor" type, but such a discussion, if at all thorough, would be too lengthy to be included at this time. Whether or not such specialized structures as the hippocampus (Ammon's horn), uncus, amygdaloid complex, and the buried hippocampus should even be considered as a part of the temporal lobe, the point of view of most clinicians,[6,15] or as belonging to the limbic lobe, the feeling of most anatomists and physiologists, is a disputed point.[53] The connections of these structures with the olfactory system, rhinencephalon, septal area, hypothalamus, thalamic nuclei, and mesencephalic reticular formation are well known, as are their connections with their contralateral counterparts.[15,54–57] Furthermore, their proximity as well as their connections with the insula, orbital cortex of the frontal lobe, and basal ganglion structures, are evident. The amygdala may play a most important part in the production of sensory seizures and automatisms.[54,58] Its medial or corticomedial nuclear group receives terminal fibers from the olfactory tract and discharges to the hypothalamus over the stria terminalis. The lateral or basolateral nuclei are intimately related with those on the opposite side through the anterior commissure, with the overlying hippocampal gyrus by association bundles, and with the anterior hypothalamic region by longitudinal association bundles and the stria terminalis.[59] Kaada has reported that stimulation of electrodes implanted in the amygdala causes arrest of movement, licking, chewing and swallowing responses, turning of the head and eyes, and searching movements,[60] and Baldwin has produced movements of the face and jaw following stimulation of the mesial amygdaloid area.[61] However, similar manifestations have also been produced by stimulation of the rhinencephalic cortex, diencephalic structures, and the temporal lobe cortex.[62,63] It is probable that a discharge starting in

any of these areas is relayed to other structures, and that the automatism and other manifestations of the fully developed attacks are postictal phenomena, dependent upon the spread of the discharge.[8,54,64] Walker has recently suggested, for instance, that some of the motor phenomena of psychomotor epilepsy may be initiated from the globus pallidus, caudate nucleus, hypothalamus, and subthalamic nucleus.[65]

The experimental and anatomic aspects of this subject are most intriguing, and should not be terminated with this brief discussion. Furthermore, these aspects are still undergoing development and modification, and will have to be reviewed more in detail at a later date.

In conclusion, we have not yet solved the problem of nomenclature of this chapter of epilepsy. The name one must choose will depend in part on whether he wishes to use a clinical or an anatomic designation. I must confess that I use both: "psychomotor" when referring to the process or syndrome as a whole, and "temporal lobe" when referring to individual aspects—illusions, hallucinations, déjà vu and other memory disturbances, and so on. I feel personally, however, that the term temporal lobe is too restricted and that many of the features of the complete attack are the result of a discharge either originating in or relayed to structures other than those confined to the anatomic bounds of the temporal lobe. With Gibbs, Gastaut, and others, I feel that psychomotor epilepsy may be regarded as a clinical entity, or perhaps a better terminology would be "clinical syndrome." If we use the term, however, we must not regard it as a disease *per se*, but as a variety of seizure pattern. If we use the term, we must do so with a thorough understanding of the anatomic and physiologic background of each manifestation of the total disorder.

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■ As regards the treatment of epilepsy, we will only say that the principal rule is to find out if the disease has an external cause – i. e., if from any part of the centripetal nerves there is an irritation acting upon the nervous centres. To prevent this irritation reaching these centres, or to destroy the cause of this irritation if it is known to exist, are the two things to be done. I must repeat that this will be found much more often than is generally supposed. Against the increased excitability of some part of the nervous centres, the best means, assuredly, are the powerful modificators of nutrition, which, I am sorry to say, are so little employed by regular practitioners – the cauterization of the back of the neck by moxas, or by the red-hot iron.

C. E. Brown-Séquard in *Lectures on the Physiology and Pathology of the Central Nervous System*, published in 1860.

Face and Jaw Movements During Epileptiform Discharge in Temporal Regions

*Maitland Baldwin, M.D., Laurence L. Frost, Ph.D. and Charles D. Wood, M.S.**

TEMPORAL LOBE SEIZURES are characterized by a particular alteration of consciousness, peculiar movements, and autonomic changes. Frequently these ictal phenomena are preceded by unusual sensations or bizarre mental experiences which serve to warn the patient that an attack is beginning.[1]

There is an ictal sequence in temporal lobe seizures, as in other varieties of focal epilepsy. Usually consciousness is altered first. This is a fascinating phenomenon and, unfortunately, the least understood of all the ictal sequences. As consciousness alters, the pupils dilate, the face changes color, and the palpebral fissures widen. The patient seems to stare. In most cases widening of the palpebral fissures is the first movement in the attack sequence. Moreover, movements of the face and jaw always occur early in the seizure and they are among the most characteristic of these strange ictal phenomena.

Of these phenomena, the most obvious and perhaps the most characteristic are masticatory movements. Magnus and coworkers[2] attributed the sucking, chewing, lip smacking, and swallowing movements to an epileptogenic lesion which is deep and mesial within the affected lobe. They emphasized that masticatory movements always occur after the fundamental alteration in consciousness.

Grimacing and winking also occur, but these are less common and perhaps less characteristic. We have observed these facial contortions in the habitual seizure patterns of several cases. They occur early in the ictal progression and seem to follow the widening of the palpebral fissures, while they precede the masticatory phenomena. A grimace is quite different from the clonic jerking of the corner of the mouth or the side of the face which may occur late in the sequence of movements. It usually occurs early in the ictal sequence, and occasionally it precedes all the clinical phenomena of the seizure.

As examples of grimacing which occurred at the beginning of a temporal

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lobe seizure, we present the following cases:

Case 1. The patient was 19 years old on admission two years ago. He is right-handed. His attacks began at the age of four years and continued despite intensive medical treatment. Each seizure began with a feeling of warmth in his face. This was soon followed by a "pulling up" of the left corner of his mouth. He was conscious at this time and frequently pointed to his mouth, saying, "Look at it. I am having one." He could not control the movement. As the movement progressed he felt a tingling in the left side of his face and objects seemed to move away from him. His consciousness altered, his head turned to the left, and he raised his left arm. Thereafter he fumbled with objects, inspected his person, or walked about. In one such attack, he dismantled the electroencephalograph connections and tore the Grass cable from its moorings. On occasion, he would continue his preictal activity in an aimless and repetitive fashion. After the attack he had difficulty in naming objects.

Pneumogram revealed a slight relative enlargement of the left temporal horn. Electrographic analysis revealed a predominant left temporal epileptogenic activity. There was some abnormality in the ipsilateral parietal and contralateral temporal regions as well.

His seizures were frequent despite medication and his social plight was desperate. Therefore exploration of the left temporal lobe was undertaken in a left osteoplastic craniotomy. Figure 1 shows the area of exploration and his habitual ictal grimace. During exploration an area for speech was outlined. On the basis of focal electrographic abnormality, reproduction of the grimace, and objective abnormality (incisural sclerosis and atrophy), the left temporal lobe was removed down to the mesial pia. The lateral extent of this removal is shown by the dotted line.

His subsequent course was benign and he has not experienced any further habitual seizures in the subsequent two years. The electroencephalographic abnormality recorded from right temporal and left parietal leads has disappeared.

Case 2. The patient was 43 years old at admission three years ago. He is right-handed. His birth was difficult, and his attacks began in his second year. Each attack was ushered in by "an indescribable feeling that things are not quite right." Thereafter his consciousness altered, his palpebral fissures widened, and his mouth pulled upward and to the right. He began to chew. Then he salivated and fumbled with his clothing. There were no postictal signs. Further analysis revealed a slight relative dilation of the right temporal horn. There was a focal epileptogenic electrographic abnormality over the right temporal region.

His attacks had continued for many years despite adequate medication. Therefore, exploration of the right temporal lobe was undertaken after an osteoplastic craniotomy. His ictal grimace and the results of exploration are shown in figure 2. Since this exploration revealed significant electrographic and objective abnormality, we removed the area anterior and mesial to the dotted line shown in figure 2. His postoperative course was benign, and he has not had any attacks for three years.

In each of these cases the attack began with facial movements, and in each case these grimaces were on the same side as the significant epileptogenic lesion. In the first case, the patient was conscious as the grimace began; in the second case, the mouth did not move until after consciousness had been altered.

In our observations of temporal lobe seizures, these ipsilateral facial movements are unusual. In table 1 are summarized the movements of face and jaw which we have observed as part of the ictal sequence in 40 cases of temporal lobe seizures which came to surgical treatment.

Because of the unique characteristics of these ipsilateral facial movements, we tried to reproduce them as part of an ictal sequence in the experimental laboratory. We hoped to evoke other movements of face and jaw as well, in order to study their form, location, and origin.

MATERIALS AND METHODS

In the laboratory seven monkeys (Macacca rheus) and two chimpanzees (Troglodytes Niger) were studied.

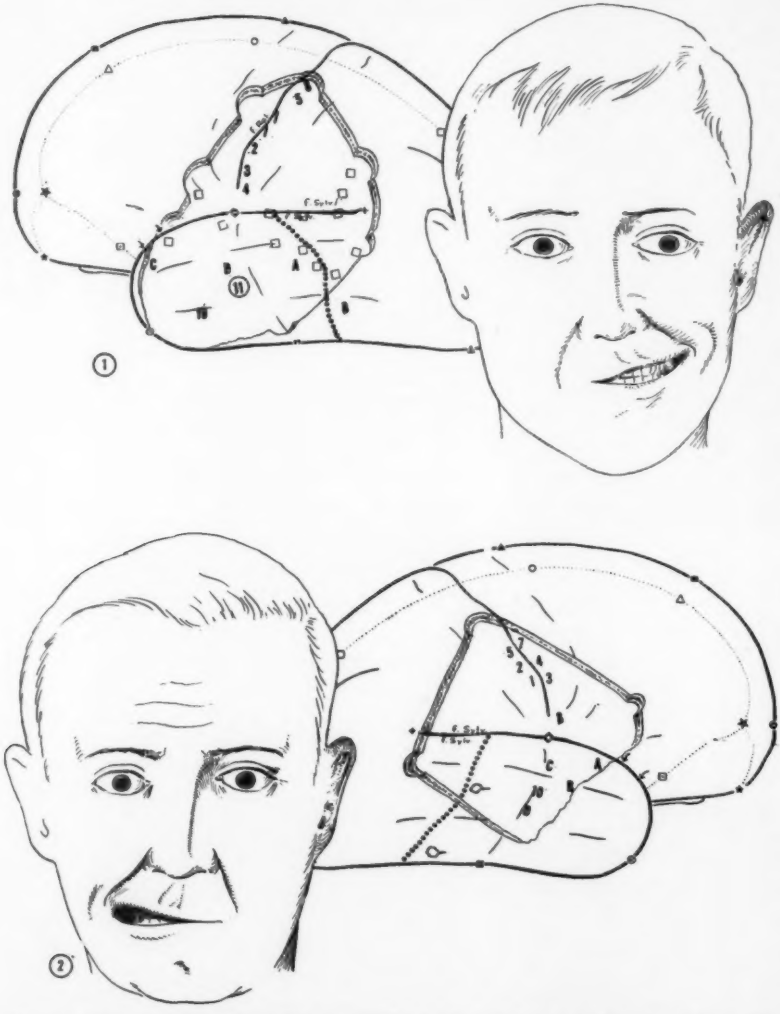


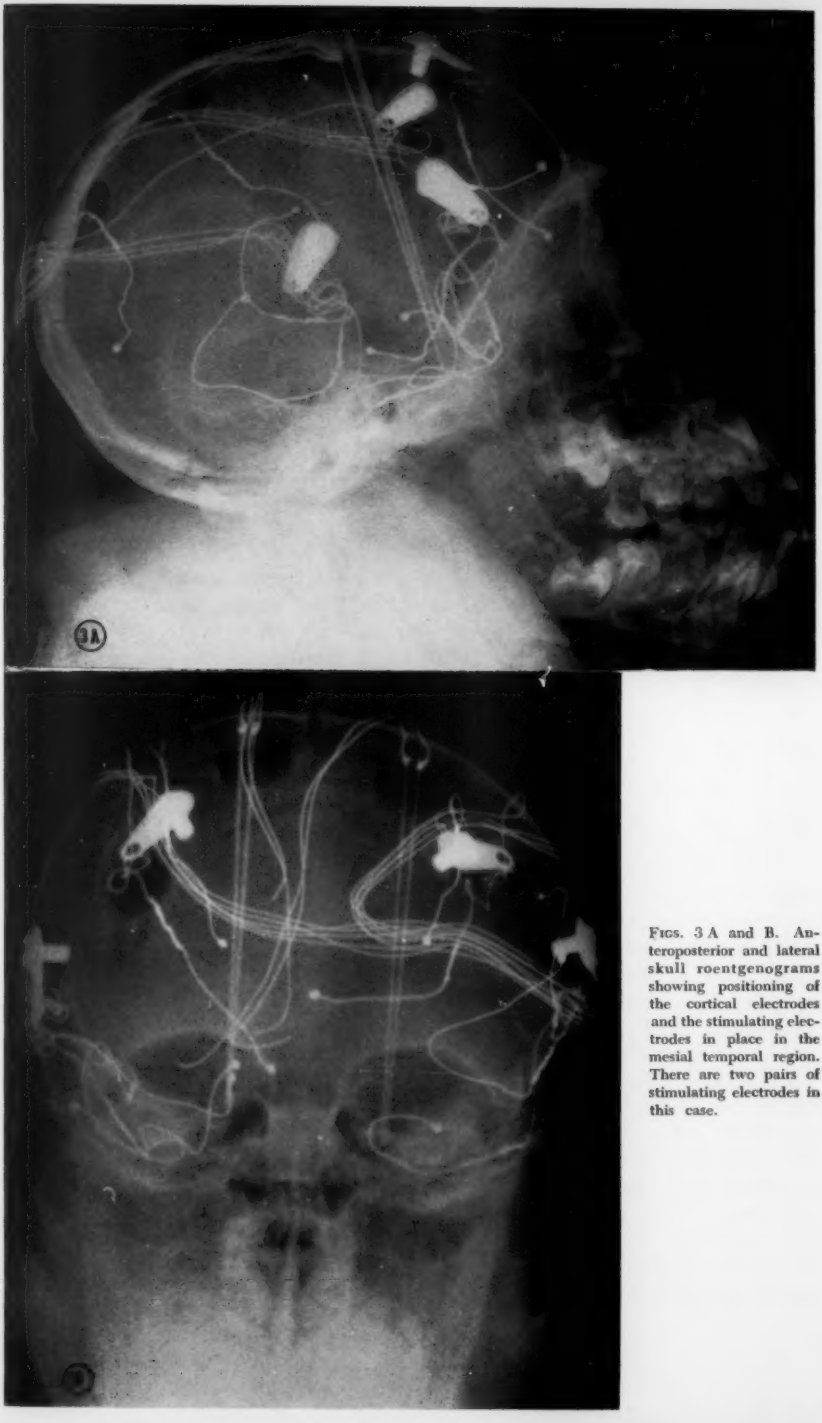
FIG. 1. Artist's conception of the initial movement of the lips and jaw in a spontaneous seizure. The brain chart outlines the craniotomy. Deep stimulation at point 11 using 8 volts reproduced this movement on the operating table.

FIG. 2. Artist's reproduction of the initial movement of the lips and jaw in a spontaneous seizure. The brain chart indicates the craniotomy. Deep stimulation at point 10 reproduced the illustrated facial movement at operation.

TABLE 1
MOVEMENTS OF THE FACE AND JAW DURING SPONTANEOUS TEMPORAL LOBE SEIZURES

Stimulation No	Side of Operation	E.E.G.	Ipsilateral	Bilateral	Contralateral	Lips	Cheek	Jaw	Tongue	Grimace	Smacking	Sucking	Chewing	Licking	Swallowing
S-67	L	L & R	X			X	X			X					
S-68	L	L ant. & mid.			X	X									
S-70	L	L ant.					X		X		X		X	X	
S-74	R	R ant.		X		X	X				X	X			
S-79	R	R ant.		X		X	X				X	X			X
S-85	R	R inf.	X			X				X					
S-92	R	R ant.		X		X					X				
S-94	R	R inf. & ant.			X	X				X					
S-95	L	L	X	X		X	X	X	X	X		X	X		X
S-96	R	R ant.	X			X	X			X	X				
S-97	L	L				X	X				X				
S-99	L	L		X		X		X	X	X	X				
S-100	L	L post.		X		X				X			X		
S-101	R	R inf.		X		X							X		
S-103	R	R		X						X					X
S-104	L	L mid.		X							X		X		
S-107	R	R inf.		X		X					X				
S-109	R	R ant.		X	X	X			X						X
S-111	L	L		X		X					X				X
S-113	R	R				X	X				X		X		X
S-115	L	L ant.				X					X		X		
S-116	R	R		X		X	X	X					X		
S-117	L	L ant.		X							X				X
S-121	L	L mid.		X			X						X		X
S-123	R	R	X	X		X				X	X		X		
S-130	R	R	X	X		X					X	X			X
S-133	R	R & L	X	X		X		X			X		X		
S-136	L	L mid. & post.		X				X					X		X
S-138	R	R inf.		X		X		X			X		X		
S-144	R	R				X		X			X		X		
S-145	L	L & R		X		X	X				X	X	X		
S-148	L	L		X		X		X			X		X		
S-17	R	R			X	X	X								
S-18	L	L ant. & post.				X									
S-19	R	L ant.		X		X					X				
S-22	L	L ant.		X		X				X	X	X			
S-24	L	L		X		X				X	X	X			
S-26	L	L											X		
S-28	R	R ant.		X		X					X	X			
S-33	R	R ant.		X							X		X		

Bipolar concentric stainless steel electrodes (diameter: 0.508 mm.; inter-electrode distance: 2 mm.) were inserted in each monkey's brain. These were directed with a Horsley-Clarke apparatus. One set was implanted on each side in every



FIGS. 3 A and B. Anteroposterior and lateral skull roentgenograms showing positioning of the cortical electrodes and the stimulating electrodes in place in the mesial temporal region. There are two pairs of stimulating electrodes in this case.

animal. Also, recording electrodes were placed over the occipital, parietal, and temporal cortices. These electrodes were insulated except at the tip, which was silvered. All connections were drawn out beneath the galea to the surface through a stab wound in the midline of the neck. The deep electrodes were fixed to the skull with an acrylic substance and the recording electrodes anchored to the bone with stainless steel wire.

All procedures were accomplished under sterile technic with light Nembutal anesthesia. All animals recovered without incident.

The two chimpanzees were used for a variety of purposes which did not interfere with the present experiments. For this experiment bipolar stainless steel electrodes were inserted in the depth of one temporal lobe. In one animal the right temporal lobe was entered and in the other animal the left temporal lobe. These apes had small heads and it was possible to modify a Horsley-Clarke stage to fit the head size. This modification was helpful, but it obviously did not provide complete accuracy since we had no coordinates. Therefore pneumograms were done in each animal. Then the tip of the right or left temporal horn was used as a guide for implantation in conjunction with the modification of the Horsley-Clarke technic. We aimed for the superomesial part of the temporal horn (figures 3A and B). The electrodes were fixed to the skull with an acrylic substance and their connections carried beneath the galea to a stab wound at the midline of the neck. Over each frontal, temporal, and parietal cortex recording electrodes were placed and their connections withdrawn beneath the galea to the neck. All procedures were done with sterile technic under light Pentothal anesthesia.

In each animal repetitive electrical stimulations were applied to the depth electrodes on one side. This stimulation was derived from a Grass stimulator and connections. In the monkeys the parameter at which an afterdischarge could be established averaged 5 volts with a 60 cycle current of 1.5 msec. duration. In the two apes an average voltage of 7.5 with the same current characteristics was used.

We recorded from the cortical electrodes with a Grass electroencephalograph before, during, and after stimulation. In the monkeys we were able to record from one set of depth electrodes as well. (We were stimulating along the other depth electrodes.) In four monkeys we stimulated along the course of the anterior commissure while we recorded from both amygdalae and the cortex.

Each stimulation was repeated several times and each preparation was stimulated on several occasions. The results of electrical stimulation were observed under light barbiturate anesthesia and while the animals were conscious and alert. The conscious preparations were photographed in color.

OBSERVATIONS

In each monkey facial movements which began with the repetitive stimulation were observed. These movements began on the side of the stimulating electrode. The most frequent movement was an eversion of the ipsilateral upper lip, followed by a pulling of both upper and lower lips toward the side of stimulus. Once an afterdischarge was recorded in the opposite depth electrodes, the facial movements became bilateral.

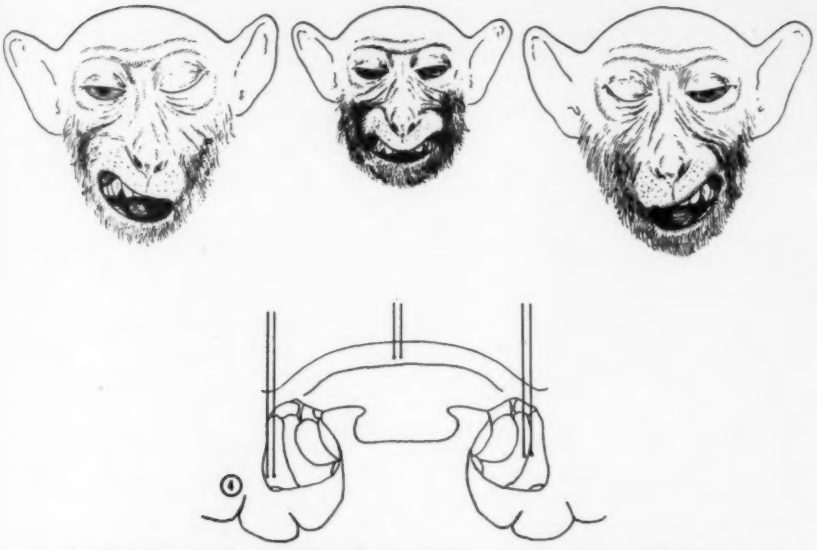


FIG. 4. The artist's conception of initial ipsilateral and bilateral facial movements which followed repetitive stimulation in the amygdalae or anterior commissure. The positions of the electrode tips relative to the sides of the movements are shown.

Coincident with the pulling of the mouth to the side of the stimulus, the ipsilateral eye would often close. Occasionally the jaw clenched and twice the mouth opened. In a few instances a prolonged afterdischarge was coincident with chewing movements.

If the monkey was conscious as the stimulus began, his face movements were soon followed by head-turning away from the side of the stimulus, and as the seizure continued, he clenched his contralateral fist after extending the arm.

There was an inspiratory arrest at the onset of each induced seizure and every continuing afterdischarge was accompanied by a tachycardia. In some instances salivation occurred.

Figure 4 illustrates the initial lip movements as they occurred during stimulation of the amygdalae and the anterior commissure. These are summarized in table 2.

The afterdischarge was first recorded in the opposite amygdala. Usually this recording was accompanied by a "flattening" of the record from the ipsilateral temporal cortex and then by the appearance of "spikes" in the tracing from the opposite temporal cortex. Actually, the repetitive stimulation of one amygdaloid complex was always followed by electrographic changes in all cortical areas from which recordings were made. The most striking change was always recorded from the parietal cortex on the side of stimulation. The coincident activity in occipital and frontal cortex was slower, with variation in amplitude. Occasionally all cortical electrodes recorded "spiking."

TABLE 2

AN ANALYSIS OF OBSERVATIONS MADE DURING ELECTRICAL STIMULATION OF THE AMYGDALA IN THE MACACA MULATTA

No.	Parameter	Location of Lesion in Amygdaloid Nucleus	Side	Conscious or Anesth.	Movements					
					Lip	Tongue	Jaw	Eyelid	Head	Autonomic
1	3-5 V	R Lateral L Basalis lateralis	R L	C C	Yes			Close		Respiration
			R	A	Yes			Close		
			L	A	Yes			Close		
2	3-5 V	R Lateral L Medialis	R L	C C	Yes Yes					Respiration
3	3-5 V	R Lateral L Lateral	R L	C A	Yes Yes			Open Open		
4	3-5V	R Lateral L Medialis	R L	C C	Yes			Close	To left	Respiration
			R	A	Yes			Close		
			L	A	Yes			Close		
5	2-3 V 4-6 V	R Medialis L Lateral	R L	A C		To left	Close Open-close	Open Open		
				A	Yes			Close		
6	4-5 V	R Medialis	R	C	Yes	To left	To left	Close	To left	
				A		To left	To left	Close	To left	
	4-7 V	L Lateral	L	C	Yes	To left	Open-close	Close		
				A			Close			
7	4-6 V	R Lateral	R	C	Yes			Close	To left	
				A	Yes		To right		To left	Tearing
	3-4 V	L Lateral basal accessory	L	C A			Open-close	Open-close	To left	salivation

Key: R: Right, L: Left, Respiration: Inspiratory Arrest.

A sample of the electroencephalographic changes following stimulation of the right amygdaloid complex in one monkey is shown in figure 5. The afterdischarge lasted from five to 15 seconds and most frequently consisted of three to five per second spikes or spike-and-wave complexes, with an amplitude varying from 150 to 250 microvolts. Occasionally during the afterdischarge the amplitude and frequency of all cortical activity were reduced. This generalized alteration in cortical potentials often continued for some seconds after the afterdischarge had ceased.

In the apes, after deep stimulation, we observed ipsilateral eversion of the lips, pulling of the face toward the stimulus, and head-turning away from it. In some instances the ipsilateral grimace became bilateral, and chewing and salivation occurred as the attack continued. As the head turned, the contralateral fist clenched and the arm extended. Occasionally the attack persisted for five minutes, but most attacks lasted from three seconds to three minutes. The animals seemed dazed after the attack subsided, and if several seizures were induced in sequence, each animal became drowsy and apathetic. This altered awareness lasted for some time after the seizures. During this period the chimpanzees did not respond to food and were negligent in responses to handling. One crawled in the corner of his cage and slept.

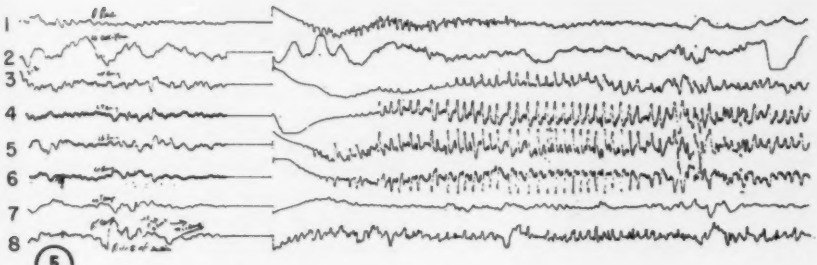


FIG. 5. Electroencephalographic tracing from cortical and amygdaloid electrodes showing an afterdischarge primarily in the left amygdala and right parietal electrodes following stimulation at 14 volts, 60 cycles/sec., 2.5 msec. duration, delivered to the right amygdaloid nucleus. Line 1 is the right parietal to indifferent; line 2, left occipital to left parietal; lines 3, 4, 5, and 6 are from depth electrodes in the left amygdala going from mesial to lateral; lines 7 and 8 are recordings from left and right temporal cortices, respectively.

The initial ipsilateral grimace is illustrated in figure 6; figure 7 illustrates contralateral head-turning and salivation.

We could not record from the subcortical structures during these stimulations. Cortical electrodes did record some of the distant effects, however. Thus, after a repetitive stimulus of 8 volts, 60 cycles per second, 2.5 msec., lasting five seconds, we recorded "spikes" from various cortical regions. The ipsilateral temporal cortex "fired" first, followed by the opposite homologue. Usually the ipsilateral parietal cortex fired next. Occasionally the parietal cortex fired as spikes appeared in the ipsilateral temporal cortex. However, ipsilateral temporal activity usually occurred one to two seconds before the ipsilateral parietal cortex fired. The amplitude of this activity was most frequently 100 to 150 microvolts and consisted of spikes at approximately ten per second. Occa-

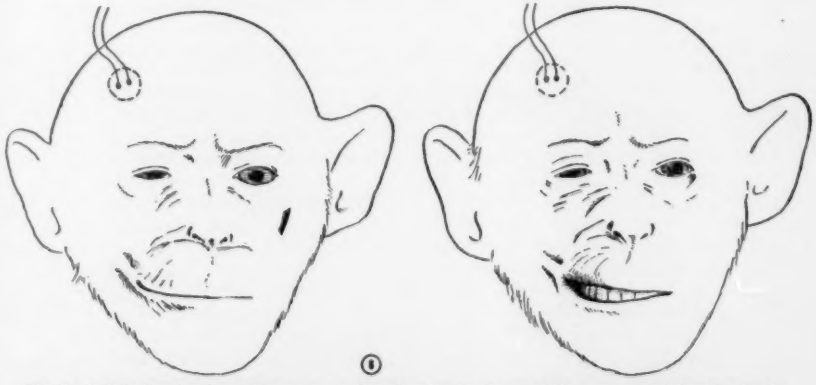


FIG. 6. Artist's drawing from 16 mm. movie film showing two stages of lip movement following stimulation of the right mesial temporal region in the chimpanzee—stimulation at 8 volts, 60 cycles/sec., 2.5 msec. duration.

FIG. 7. Artist's tracing from a 16 mm. movie film of a chimpanzee following stimulation at 8 volts, 60 cycles/sec., 2.5 msec. duration, delivered to the right mesial temporal region, showing salivation.

sionally the frontal cortical electrodes recorded spiking after or during the temporal firing. If the parietal cortex fired, this firing continued longer than that recorded from other areas. Figure 8 is a sample tracing recorded after stimulation in the mesial temporal region of the right side.

When the animal's awareness was altered, the electrogram showed slow activity. During the intervals between experiments when the apes were caged, no unusual behavior was observed.

SUMMARY

In certain primates, repetitive stimulation of one mesial temporal region is followed by ipsilateral facial movements. This intense electrical stimulation induces an afterdischarge which can be recorded from the opposite mesial temporal structures. The further electrographic effects of the afterdischarge can also be recorded from temporal, parietal, and frontal cortex. It can be established easily at low voltages, and once established it continues for a time. During

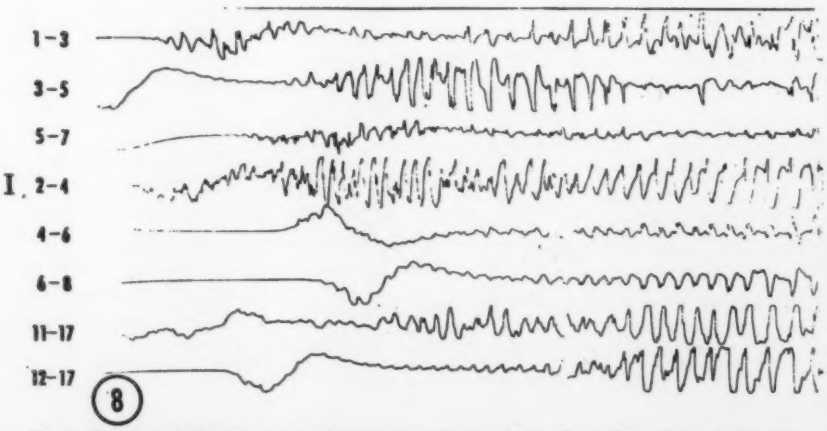


FIG. 8. Electroencephalographic tracing showing seizure discharges after electrical stimulation in the right mesial temporal region of the chimpanzee. Electrode placements: *Subdural:* (1) left temporal pole, (2) right mesial temporal, (3) left lateral temporal, (4) right temporal pole, (5) left frontal, (6) right lateral temporal, (7) left parietal, (8) right frontal. *Skin:* (11) left anterior temporal, (12) right anterior temporal, (17) vertex.

this period consciousness is altered, the face, jaw, head, and contralateral upper extremity move, and the animal may salivate or lacrimate. Occasionally the ipsilateral grimace occurs before consciousness is altered. There is usually a concomitant tachycardia and an initial inspiratory arrest.

In certain patients, focal epileptiform activity is followed by ipsilateral facial movements. This has been observed in six cases in which the epileptogenic lesion was localized in the mesial temporal region. These ipsilateral movements occurred as initial phenomena in the habitual ictal sequence of these patients. However, facial movements which occur in the ictal progression of a temporal lobe sequence are more commonly bilateral. When the initial movements of the face are one-sided, they may occur on the same side as the significant epileptogenic lesion which lies deep within the mesial temporal region.

These studies have been accomplished with the technical assistance of Miss Shirley Lewis, R.N.

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■ The persistent spasmodic contractions, due to a mechanical injury to certain parts of the nervous centres, are always curious, but never so much so as when they result from some irritation of a part like the auditory nerve, which we were accustomed to consider simply as a nerve of sense. M. Flourens has found that the section of the semicircular canals, in certain animals, is followed by a strange disorder of movements, and sometimes by a rotation (Circus movement). I have ascertained that the phenomena observed in these experiments do not depend on the section of these canals, as this operation may not cause these phenomena, but that they are the results of an irritation of the auditory nerve, from the drawing upon it by the membranous semicircular canals at the time we divide them. In frogs and in mammals, the direct irritation of the auditory nerve is followed by the most interesting phenomena. It is well known that in frogs the peripheric extremity of this nerve is inclosed in a bag containing carbonate of lime; as soon as this bag is laid bare and slightly touched, and still more if it is punctured with a needle or a bistoury, the anterior limb, on the opposite side, is thrown into a state of slight convulsion, and kept almost constantly in a spasmodic pronation; and almost at every attempt to move forwards the animal turns round on the side injured. As long as it lives (many days, or even many months), these phenomena may be observed, although not quite so marked as immediately after the injury, or after the first twenty-four hours. In mammals, the least puncture of the auditory nerve causes rolling, just as after the irritation of the processus cerebelli ad pontem; violent convulsions then occur in the eyes, the face, and many muscles of the neck and chest. The doctrine that the nerves of the higher senses are not endowed with general sensibility (i. e., are not able to cause pain) seems not to be true with regard to the acoustic nerve; at least, the signs of pain given after an irritation of this pretended nerve are often as great as those observed after an irritation of the trunk of the trigeminal nerve.

C. E. Brown-Séquard in *Lectures on the Physiology and Pathology of the Central Nervous System*, published in 1860.

Laughter in Epilepsy

Ralph Druckman, M.D. and Dora Chao, M.D.

DISORDERS OF LAUGHTER in the form of forced laughter are widely recognized as occurring in organic brain syndromes such as pseudobulbar palsy, generalized cerebral arteriosclerosis, amyotrophic lateral sclerosis, and disseminated sclerosis. Similar uncontrollable outbursts of laughter have also been reported following prefrontal lobotomy.[1] It is less well known that laughter may be a manifestation of epilepsy. Féré[2] first reported epileptic laughter in 1898, and a number of scattered reports have since appeared.[3–12] There does not seem to be any consistent pattern to the clinical picture of the patients with laughter as an epileptic manifestation. The electroencephalographic findings in these patients have only rarely been reported[11] and they show various abnormalities.

In the course of study of massive spasms,[12] the occasional occurrence of laughter as part of the seizure pattern seemed noteworthy. These patients and others with convulsive laughter are the subject of this report.

CASE REPORTS

Case 1. A Latin American infant was born at home with the aid of a midwife and was apparently well until two or three days of age when she began having seizures. These were characterized by flexion of arms on the chest and bending of the body in the middle, with the child remaining rigid in this position for a few seconds. The frequency of these seizures varied from ten to 30 daily. A brother had died at three months of age with seizures.

When first seen in December 1949 at the age of ten months, the fontanels were closed and the child was markedly retarded. She was unable to sit or hold up her head. The tone in the lower limbs was increased and bilateral sustained ankle clonus was present.

A previous electroencephalogram in June 1949, at four months of age, revealed continuous high voltage slow activity ranging from 1½ to 5 per second, with some asymmetry between sides. A further examination in December 1949 revealed generalized spiking and sharp wave activity occurring asynchronously in all leads. This was interpreted as indicating diffuse brain damage.

The child was given a number of medications and had fewer seizures. At two years and eight months the child was estimated to have a developmental level of one to two months; she cried continuously and kept her head hyperextended.

In March 1952 at the age of four years, the patient had seizures consisting of drawing up of the legs for a second or two. In July 1952 it was noted that the patient still had three to four attacks daily, in which she drew up her right leg or all of her limbs for a second or so, burst into laughter momentarily, and then the episode was over. The head and eyes might turn to the right in these attacks.

The patient was last seen in October 1953 at four years and eight months of age, and she was still having one or two attacks daily. No note was made regarding laughter. Spastic

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quadriplegia and severe mental retardation persisted. She was then placed in a home for handicapped children.

Case 2. An infant was born at term after an easy four-hour labor. The child breathed and cried spontaneously. At about ten days of age the infant developed diarrhea. The stools were first loose yellow and later watery green. This persisted to six weeks of age, when Terramycin was given and the diarrhea improved. At six weeks the child developed spasms consisting of momentary jerking of all limbs. The pattern of seizures changed after a few weeks so that the attack consisted of extension of the lower limbs, flexion of the upper limbs, head, and trunk. Medication reduced the frequency and severity of seizures.

At 13 months of age, when first seen at the clinic in November 1952, the child could hold her head erect and sit with support. She was hypotonic. The anterior fontanel was closed and the head circumference was 42 cm.

An electroencephalogram at 14 months of age revealed high voltage slow spikes, sharp waves, and 1½ to 6 per second waves in the occipital leads with reflection in all other leads. There was also some high voltage 3 to 10 per second activity in all leads. A clinical seizure occurred, with sudden flexion of arms and legs and of the head on the chest. The electrographic aspect of this attack consisted of cessation of the high voltage epileptiform activity, a burst of 10 to 11 per second waves, followed by continuous 6 to 7 per second activity in the anterior leads and flattening of activity in the occipital leads. About 20 seconds after the seizure the continuous high voltage abnormality reappeared.

The seizures gradually lessened in number until at 16 months the patient had spells just three times daily. These usually occurred right after sleep or a nap. The arms and legs would stiffen for a few seconds and the child would laugh and giggle. No further note about laughter was made after this clinic visit in January 1953.

The attacks lessened, but with three bouts of respiratory infection the child had more attacks with pulling of the body forward and throwing of the head forward. At two years of age the child still had mild seizures. The child startled and jerked upon loud noises. At two and a half years the seizures ceased, only to return at three years of age, still "jackknife" in type. At that time the child could move around by herself on the bed. The head circumference was 43.2 cm. The tendon reflexes could not be elicited. A repeat electroencephalogram at three years and five months showed continuous high voltage 14 to 15 per second activity, mixed with very high voltage 7 to 9 per second activity and with scattered high voltage 3 to 4 per second transients anteriorly.

At four and one-third years of age when last seen, the child still had about one jackknife seizure weekly, precipitated by sudden wakening. She could now hold up her head, smile, follow objects with her eyes, and roll over from back to stomach.

Case 3. This child's birth was at term after a few hours of labor. There was no neonatal difficulty. He was normal until seven weeks of age when he was injured in an auto accident; a tire blew out and the car whirled around several times. The patient was found lying still with a swelling of the right side of the head. It could not be determined with certainty whether the child had lost consciousness. Roentgenography revealed a skull fracture. After three weeks in the hospital the child was dismissed home. He had a left hemiplegia and just lay in bed all the time. One month after leaving the hospital, he began to have seizures characterized by extension of the left arm and flexion of the legs and of the head. These attacks usually occurred upon awakening and in rapid succession. He might then stare as if frightened or sometimes laugh "unnaturally." He might also cry after a seizure. These attacks occurred about 12 times daily and were reduced in frequency by medication with Dilantin and phenobarbital. This was the only mention of laughter.

At six months an exploratory craniotomy was performed and a region of cerebral atrophy was found. The child began to hold his head up at seven to eight months, but was unable to sit up when seen at one year of age in March 1953. He was uninterested in things going on about him and had a left spastic hemiparesis. He continuously beat at the bed with his right hand. Both plantar responses were extensor and the tendon reflexes were brisker on the right side. An electroencephalogram at that time showed frontal dominant slow spike and wave complexes awake, with long bursts of generalized polyrhythmic fast spike and slow wave discharges in sleep and

focal slow spike and slow wave activity in the right occipital region.

Mebaral and Gemonil were given and the attacks became more focal in nature, with jerking of the left lower limb. Jackknife seizures continued to 22 months of age and then ceased. He still had four to five attacks daily in which his left arm was thrown out once. Examination at three years and eight months showed a left spastic hemiplegia with left hemiatrophy. Tone and tendon reflex activity were also increased on the right side, but not nearly to the same degree as on the left. No further laughter had occurred. A repeated electrogram at this time revealed a very active focus of slow spike and rapid multiple spike and slow spike and wave activity in the right superior central region. A moderately high voltage slow dysrhythmia was also present.

Case 4. The gestation period of this child was full term and normal, except for occasional albuminuria in the third trimester. This was the mother's first pregnancy and she was deeply sedated with many injections during the entire eight-hour labor. The child was blue after birth and was kept in a head-down position in her crib for the first six days of life. The baby fed well, but slept excessively for the first three months.

At four months of age the infant developed seizures occurring about five to six times daily. She would lunge her head forward and at the same time throw up her arms and legs. An electroencephalogram at the age of seven months showed numerous polyrhythmic asynchronous multiple spike discharges interpreted as fitting the pattern seen in "infantile spasms." Medication with phenobarbital and Dilantin was begun. At eight months the baby began to arch her back unusually and preferred to have the head arched back as well. This continued to ten months. The seizures continued at a frequency between ten and 200 daily, with the seizures most likely to occur just after awakening. For a few weeks she laughed prior to the onset of a seizure, but then would become irritable or cry before the attack and might cry directly after, too.

At four and a half months the child began to hold her head up from the bed. At 11 months she could sit alone for three minutes, but still without full head control. She then also began reaching for toys, and she could laugh naturally.

The child's mother had had seizures, both with and without fever, between three and five years of age. Several of the mother's siblings had febrile seizures in infancy and childhood.

On examination at age one year in April 1954, the child was retarded but showed no other abnormal findings. An electroencephalogram at that age showed the diffusely abnormal pattern of "hypsarhythmia."

Gemonil was added to the medication and the child had fewer seizures. At 17 months the child was having about three spasms weekly, usually on awakening in the morning. Her eyes would roll back and her mouth pull to the right, her arms abducted, and the lower limbs would flex on the trunk and then quiver. After remaining in this position for one to two minutes, she would stop quivering and then often laugh for a minute or so. She laughed more after single spasms than after longer series of spasms. The mother thought the laughter was forced and resembled that seen in children before they cry; the patient did not go on to cry. The laughter persisted for a few weeks and then disappeared. Another electrogram at 17 months of age revealed more normal frequencies and periods of relatively normal activity.

At 22 months the child was having four to eight spasms daily, some of them in series. In these her eyes rolled back, her face was brought to her toes, and her arms were thrown out at the sides; she would turn blue. The lesser attacks lasted only a fraction of a second, but she might remain in one up to three or four minutes. She would sleep after the more severe attacks. She was also having akinetic and tonic extensor attacks. She had begun to stand and walk with support and could distinguish her father and mother.

Case 5. The pregnancy was complicated by continuous vomiting with severe headache between one and a half and three and a half months of gestation. The child was born at term after a labor lasting five hours. Birth weight was six pounds, four ounces and the child was kept in an "air lock" for 12 hours. She fed well, achieved head control, and turned over at three months. She played with a rattle and laughed at three months.

The patient was considered perfectly normal until six months of age. Then after a feeding the child seemed unusually quiet and her mother found that she was not breathing. Her lips were blue and she was limp. After about

two minutes the patient regained consciousness. The next morning the child had a further attack of limpness and cessation of respiration lasting about half a minute.

One week later while the mother was holding the child, the child suddenly drew up into a ball with flexion of limbs and trunk. The child then relaxed but went on to have five more such massive flexor spasms in rapid succession. She then broke out in a sweat. The child continued to have three to 15 such attacks daily. She became cross and irritable and stopped turning over in bed.

A paternal aunt and great aunt had seizures in childhood.

On examination at age six months the child displayed little interest in her surroundings and did not handle proffered objects. A faint angiomatous area about 1 centimeter in diameter was present over the bridge of her nose.

An electroencephalogram at that time showed the typical pattern of "hypsarhythmia." Medication with Dilantin and Gemonil was begun.

The child had slightly fewer seizures, which tended to occur on awakening or on going to sleep. At eight months of age she had an extensor spasm of the arms and legs, followed by two or three minutes of laughing sufficiently violent, prolonged, and unusual to have alarmed the mother. At this time a left hemiparesis was noted.

She continued having spasms which responded only slightly to changes in medication. She would have sudden extension of her limbs into a spread-eagle position and then laugh a few times. The laughter might also precede the attack. The child did not laugh at times other than when she had attacks. She might smile without laughing after the attacks. The mother thought the sound was like normal laughter.

The attacks continued, more when fretful or when feeding. When a spoon touched her lips she might draw up all four limbs, quiver, and then stretch out. She continued irritable.

At 11 months of age she still had massive flexor attacks and was irritable. She was able to follow people about with her eyes. Mild laughter still accompanied her attacks. At 12 months another electrogram was similar to the previous one, with the additional finding of an active focus of spike and multiple spike and slow wave discharge in the left temporal region.

At 13 months of age she was having 15 to 20 seizures daily in which there was nodding of the head forward, followed by clenching of the fists. Her eyes rolled up and she did not breathe in the harder spells. She had sudden isolated general jerks "as though stuck by a pin." She was not able to sit up or grasp objects placed in her hands.

Case 6. This child was the result of the third gestation and delivery was an estimated ten days beyond the expected date of confinement. Delivery was uneventful, with an eight-hour labor and with low outlet forceps. The infant was cyanotic for one hour after delivery and resuscitation was given. A fever of 102° F. developed at two days of age and penicillin was administered. He fed and sucked well to four days of age and then failed to suck and did not feed well. The mother also noted the absence of the Moro reflex at that time.

At four and a half months of age seizures were noted to occur. The infant drew up his arms and legs and his head fell forward. He remained stiffly in this position for a few seconds and then relaxed. He might have such spasms every 20 seconds or more over periods as long as ten minutes. At six and a half months the seizures decreased in frequency so that they were occurring just once every two weeks. At ten months the seizures changed slightly in that the head would turn to the left and down before the spasm. The infant did not use his left upper limb nearly so well as the right from birth. He held his head up at three weeks, rolled over at eight months, and sat alone at 11 months.

There was no significant family history of disease.

On examination at 11 months in November 1955, the patient was noted to have flattening of the skull in the right parietooccipital region. Nystagmoid movements were present. He showed much less spontaneous movement of his left limbs, with increased tone and tendon reflexes on the left side. There was a left homonymous hemianopia to threat stimuli.

At 12 months he began to have a series of attacks daily within an hour after lunch, with turning of his head and eyes to the left and with flexion of his head and all four limbs. After about two seconds he would relax, only to have a further spasm in five to 15 seconds. There were about 15 spasms per series and he cried a little after each spasm. Some attacks also occurred at night and in one of these noc-

turnal attacks he had laughed after the last three to four spasms in the series. His mother would have thought the laughter normal except for its association with a spasm. He seemed to smile with this laughter. This infant was also capable of laughing upon appropriate stimulation at other times.

Electroencephalograms at ten and again at 11 months of age showed a very active focus of slow, sharp wave and spike discharge in the right parietooccipital region. Sleep recording revealed generalized abnormality in the form of random multiple spike discharges and sharp and slow wave activity, with poor correlation between homologous leads.

At 14 months he was having a series of attacks about every five days. There were about 30 spasms per series. He had had no further laughter in association with his spasms. He was able to sit unsupported and was trying to stand with support.

Case 7. This child was born after a pregnancy which was uneventful except for cramping upper abdominal pain and a moderately severe urinary tract infection in the last few weeks of pregnancy. Labor was initiated by rupture of the membranes and lasted 36 hours. The immediate neonatal condition is said to have been good. At three days of age the patient began to have spells which were not further described, except that they required that the child be placed in oxygen intermittently for two weeks.

When taken home from the hospital at two weeks, the parents noted that he had spasms characterized by elevation of the arms, dropping of the head, and kicking out of the legs. These occurred in runs of ten to 12 at a time and totaled about 50 per day. At nine months of age he had another type of attack in which his arms flexed and were held stiffly, his legs extended, and his face turned pale. "Goose pimples" appeared and respiration was slow and shallow.

When first seen at nine months of age in August 1955, the child had still not held up his head spontaneously and did not seem to recognize his parents.

On examination at nine months, the head was flattened anteroposteriorly and he was only able to hold his head up for a few seconds. He cooed and drooled. Skull films were normal. An electroencephalogram showed the pattern of hypsarhythmia.

Medication with Dilantin and Gemonil was begun and there was some reduction in the number of attacks. At 12 months of age, he had brief laughter following series of massive spasms. The parents thought this resembled normal laughter. He continued having laughter with his massive spasms. At 14 months of age he was having three to four massive flexor spasms in series on arising in the morning, laughing once or twice after the series. Occasionally he laughed on arising in the morning in the absence of any spasm. His mother was never able to make him laugh by stimulating him during the day, although he rarely laughed spontaneously. At 14 months he was still unable to sit up unassisted, did not chew his food, and showed much repetitive rocking activity.

Case 8. This child was the result of his mother's third pregnancy. The first pregnancy terminated in a premature delivery at six and a half months and the neonate lived only 11 hours. The second pregnancy also resulted in a premature delivery at seven and a half months, with the child living and well.

During her pregnancy with this patient the mother bled very frequently, the amount varying from a few drops to gushing. As a result she spent most of this pregnancy in bed. At seven months of gestation the membranes ruptured and four days later this child was delivered. He weighed three pounds at birth and was kept in an incubator for three months.

The child had feeding difficulty. At seven months of age a diagnosis of cerebral palsy was made. He held his head up at 18 months, sat up at four years, and crawled at about seven years. He began saying single words at two years and sentences at four years. Bowel and bladder control were established at three years.

At two years of age he had his first seizure; it lasted about 24 hours and was finally controlled by ether anesthesia. He had a series of short seizures a week later. Three months later he had a further bout of status over 24 hours, which was controlled by phenobarbital. The convulsions began with twitching of the right cheek, then spread to the right angle of the mouth, and then to the right upper limb before becoming generalized. Dilantin was begun, maintained for two years, and then stopped.

At six years he began to awaken at night and laugh without apparent cause. At seven years

of age he developed a fever of 105° to 106° F. with measles, and convulsed over a 14-hour period. He was in an oxygen tent for five days. After that his mother found that he was very hyperactive. He would laugh "hysterically" at anything. He also awakened at night, laughing for no reason. During the laughing spells the patient would look around him and not seem to realize what he was doing.

On examination at seven years of age in October 1951, he was found to have nystagmus, generalized weakness and increased tone, hyperactive tendon reflexes, and ankle clonus.

An electroencephalogram at that time showed moderate slowing, with a discharging spike focus in the right upper central region and a questionable spike focus in the right temporal region.

Dilantin was started again and the child had no further seizures or laughing spells for several weeks, when he again began to laugh at night without apparent cause. Dormison was begun at bedtime and he slept without laughing or awakening. He continued to have periods of silly giggling and laughter during the day.

He had occasional generalized seizures and his eyesight failed. He also had occasional right-sided jerkings. An intelligence test gave a formal score of 77, but it was thought that his real capacity was higher.

He was last seen at ten years and seven months of age, at which time he was having episodes at 5:00 a.m. in which he laughed uncontrollably, became clammy and cold, swallowed and rolled his eyes up. He also waved his hands and his body stiffened. These attacks lasted 20 minutes, following which the patient returned to sleep. Examination at that time revealed a spastic child with more involvement of the lower limbs. Nystagmus was no longer present.

Case 9. This patient was born at full term after an uneventful pregnancy. Labor lasted one hour and gas anesthesia was given. The baby seemed normal at birth. He raised his head at three months and walked at nine months. He had "intestinal flu" at two months of age.

At six months of age he began to have head-nodding attacks. An electroencephalogram was said to have been abnormal. Several drugs were prescribed, and Mebaral led to improvement for a two-month period. The head-nodding then reappeared and occasionally the right side of the body jerked a few times after the nodding was over.

At ten months of age he began having seizures in which an odd grin would come over his face; he would then nod his head and hold onto things with his left hand while the right limbs seemed to lose control. These episodes lasted about a half-minute and then the patient appeared normal.

At one year of age he began having attacks in which he suddenly fell to the floor, remained lying flat for a few seconds, and then got up. He then dropped his head onto his chest and appeared dazed for a few seconds. At other times he might laugh out loud at this stage. The whole episode was of one to two minutes' duration.

A maternal uncle was deaf and dumb and a paternal great uncle had seizures in childhood.

On examination when first seen at 15 months in March 1954, the only abnormality noted in the nervous system was a more active knee jerk on the right than on the left. He also had multiple small areas of depigmentation over his skin. An electroencephalogram at that time showed multiple sources of independent focal discharge in both hemispheres while awake. Massive generalized multiple spike discharges were seen in sleep.

Various medications were given, but the head-nodding attacks continued. No further note of laughter was made until two years of age, when it was remarked that the mother regarded as seizures not only the head-nodding attacks but also a brief odd smile or laugh. At two years and three months a repeat electroencephalogram revealed a very active slow spike focus in the left anterior parietal region. There was little evidence of an organized alpha rhythm.

When last seen at two years and seven months he was having seizures in which he would grin and drop his head to his chest momentarily. He might not have any attacks for two weeks and might then have one to two in one day. His development of speech continued, but slowly.

Case 10. The mother of this patient developed sudden profuse hemorrhage at nine months' gestation and the child was therefore delivered by cesarean section. The birth weight was four pounds. He was noted to be cyanotic at birth. He was weak and had to be fed with

a dropper. A supernumerary thumb was present on the left thumb and was removed later.

Clumsiness and smallness of the left side of the body was first noted at six months of age and this disability became more obvious. The boy was hypersensitive to touch, more so on the left side. He sat alone at six months, spoke his first meaningful word at seven to eight months, walked at eight months, and was talking sentences at two years. He attained bladder control by two years. Asthma was noted soon after birth.

At the age of six months the child began to have episodes in which he suddenly screamed as if in pain and held his head. After this, in about a minute, he would fall asleep and often remain asleep and unresponsive for several hours. This type of attack continued to the age of seven years when the patient was first seen. Their frequency was about once or twice weekly.

As he grew older the attacks could be described in more subjective detail. For an hour or so before an attack, the mother thought the child was less active than usual and that his eyes appeared blurred. The patient himself first noted epigastric pain and, soon after, sudden bifrontal severe pounding headache. The headache lasted about one minute. Vomiting might accompany this phase. If the attack were severe, the child might have giggling without apparent provocation. The patient stated that he did not feel that anything was funny at the time but that he could not prevent the giggling. With the initial headache he might feel dizzy and things seemed to go around. At times he had tingling of the fingers of the right hand and also about his chin. He might feel hot and cold alternately several times before losing consciousness. He also might see objects like pies or gasoline or alcohol and even smell them, although realizing at the time that these things were not really present. He sometimes became angry during this period.

The headache and associated phenomena could occur together and lasted about one minute before he fell into a deep sleep in which he was unresponsive. He usually slept about two to three hours, but on one occasion he was unresponsive for 24 hours. During this phase he might run a high fever, up to 104° F. rectally. He also perspired a great deal and sometimes had great difficulty in breathing, much as in his asthmatic attacks. With the fever he might have goose pimples. During the sleep phase the child was limp and no convulsive movements occurred, although he occasionally was incontinent of urine.

For several hours after awakening he might have feelings of warmth or chilliness. Otherwise he felt perfectly well and like a new boy. Precipitating factors included punishment, bumping his head, consuming too large a volume of food, or chewing solid foods.

Aside from the sleeping spells, other attacks occurred several times daily in which epigastric pain appeared and lasted as long as 30 minutes. His head might hurt for the same length of time. With these discomforts he sometimes became nervous and irritable but did not lose consciousness.

Ever since the mother could remember, the child had giggling spells occurring two to three times daily and lasting about five minutes. This laughter was not related to anything funny and usually came on spontaneously. The child could stop the giggling in these isolated episodes.

On examination at seven years, a bruit was heard over both eyes synchronous with the pulse. Intelligence was estimated as superior. Moderate smallness of the left limbs was present along with very slight weakness. Tone and coordination were unimpaired. Reflexes were equal and active with flexor plantar responses. No abnormality was detected in sensation. Gait was abnormal because of shortening of the left lower limb.

Mebaral reduced the number of seizures to one weekly. The addition of Dilantin to his medication further reduced the frequency and severity of his seizures.

An electroencephalogram at seven years of age revealed 14 and 6 per second positive spikes in sleep.

Case 11. This patient was born at term after a four-hour labor and weighed six pounds, nine ounces. Few details are available regarding the immediate neonatal condition, but 45 minutes after delivery the father was told that the infant had collapse of one section of his lung. The patient was given five blood transfusions. He was maintained in an air lock for one week and then stayed in the premature nursery for an additional 16 days before going home.

The infant fed well and slept well but was very quiet. He raised his head at two months,

sat alone at eight months, reached for toys at one year, walked at 14 months, and began saying single words at two and a half years but stopped again one month after he had started. He had a red spot on the top of his head which grew rapidly to the size of an egg at three months of age. Radium treatment was given and this nevus disappeared by five months of age. Another red nevus in the groin was treated but did not disappear.

The child was constantly on the go from the time that he was able to get around by himself. He liked rocking himself and jumping up and down endlessly. His coordination of hands and feet always was poor.

The father of the patient had scleroderma for some 20 years. One maternal great uncle was described as very dull and peculiar.

When first seen at three years of age, the chief complaints were of laughing spells of two and a half months' duration and inability to talk. The child had had about 20 of the laughing spells in all. He would go to bed at 9:00 p.m. and at about 1:30 a.m. his parents would be awakened by his laughing out loud. They would run to his room and find him in a dazed state. He continued to laugh in an uncontrollable fashion for 30 minutes and then stopped, remaining awake for two or three hours before falling back to sleep. His face would become flushed in these episodes. The attacks first occurred at monthly intervals, then weekly, and finally every night.

One and one-half months after onset the patient was given phenobarbital which reduced the number of attacks, but he became irritable while receiving this drug so the medication was discontinued.

Examination revealed a retarded hyperactive three year old boy. He showed adductor and tendo Achilles tightness and tended to tip-toe when walking. His coordination was poor and he was clumsy in running and climbing. He jabbered but did not speak intelligently. Internal strabismus and a high palate were noted. A faint red hemangioma was present in the right groin.

An electroencephalogram had been made at 19 months of age and showed some high voltage fast activity in sleep. This was considered of questionable significance. The waking record was normal. A repeat study was made at three years of age and revealed a poorly organized and moderately slow waking record. In sleep there was excessive high voltage fast activity.

He was started on Dilantin, Mebaral, and Thorazine, and on the first return visit he was much improved, being quieter during the day and having no further episodes of laughter. Mebaral and Thorazine were stopped and he continued to do well. He had one further episode of laughter at night two months later and then none for a year. Then he began to have episodes in which he would sit down, giggle or laugh with his eyes set and his fingers playing about his lips. He did not respond to calling during the five or so minutes of the attack. Most of the attacks were precipitated by being upset or frightened, although they might come on without precipitants. These attacks averaged two to three daily. His mother could occasionally stop an attack by shaking or calling him. He also had one further laughing attack at 2:00 a.m. His development and general interest were improving. The dosage of Dilantin was increased.

COMMENT

The epileptiform character of the laughter in these patients is indicated by the absence of any external precipitants, by the nature of the laughter, by concomitant epileptiform manifestations, and by the response to anticonvulsant medication. The laughter in these patients varied from violent laughing lasting about two minutes to mere grinning. It differed from normal laughter in that there was no contagious quality to it. Some of the parents referred to the laughter as "unnatural." In case 10, the boy indicated that he did not feel that anything was funny at the time he was laughing and that he did not feel particularly happy at that time. At least in this patient there was no affective component to the laughter.

The commonest associated epileptiform manifestation in this group of patients was the type of seizure called massive spasms. Case 9 had head-nodding attacks together with his laughter. Such head-nodding attacks are often as-

sociated with massive spasms, although they were not in this particular patient. Case 10 had a classic pattern of "diencephalic autonomic epilepsy,"[13,14] together with his laughter.

The response of the laughing attacks to anticonvulsant medication is strong evidence in support of the thesis that these attacks are epileptic. The effectiveness of Dilantin, even when used alone, is particularly important because Dilantin has practically no soporific effect.

The convulsive laughter presented in various ways. In some of these patients it occurred only in immediate association with massive spasms or with the manifestations of diencephalic autonomic epilepsy. In other patients the laughter was sometimes associated with massive spasms, but at other times might occur alone and without any obvious external precipitant. In case 11 laughter and impaired consciousness were the only manifestations of epilepsy. There is thus a gradual transition from attacks where laughter is only one of many disturbances in a seizure to episodes in which laughter is almost the only epileptiform manifestation.

It is useful to postulate the existence of a center for laughter, even if this refers only to the system of nerve cells which coordinates the movements of the mouth, face, larynx, and diaphragm in the production of laughter. The number of postmortem examinations in patients with convulsive laughter is small. Dott[8] reported one such patient who had a large astrocytoma involving the floor of the hypothalamus. List and Bebin[10] recently reported a similar patient who had a hamartoma of the hypothalamus as the underlying cause of the attacks and of the associated precocious puberty. Martin[9] has also reported a patient with what was possibly convulsive laughter who had a lesion involving the hypothalamus.

In addition to this autopsy evidence there is some information to be gained from stimulation studies. Foerster and Gagel[15] were able to provoke outbursts of laughter in a patient operated upon under local anesthesia. This patient had a papilloma of the choroid plexus of the third ventricle. Each time that the surgeons swabbed the blood from the floor of the third ventricle, they were able to provoke outbursts of laughter.

This evidence derived from pathologic and physiologic studies indicates that the mechanism for laughter is at least in part in the hypothalamic region. The clinical data presented here tend to support this localization, for the massive spasm type of seizure is thought to have a brainstem origin and this type of seizure was the commonest accompaniment of the convulsive laughter. The pattern of attack in the patient (case 10) with temperature changes, pallor, sleepiness, and goose flesh in association with the laughter is characteristic of what has been called "diencephalic autonomic epilepsy." The electrographic pattern of 14 and 6 per second positive spikes in this latter patient may also be taken as supportive evidence of a diencephalic localization of the site of abnormal discharge.[16]

All of the patients with laughter in epilepsy showed some evidence of brain injury upon examination. The abnormalities included mental impairment, hemiparesis, hemiatrophy, and spasticity. In this series none of the patients provided any evidence from physical examination which would support the hypothesis of a large hypothalamic lesion.

The electroencephalograms of these patients showed much variation, but in general supported the clinical evidence of some brain damage. The patients with massive spasms often showed hypsarhythmia. The others demonstrated slow dysrhythmias, multiple foci of spike discharge, and one patient showed the 14 and 6 per second positive spike pattern. Gibbs and Gibbs[11] have reported the electrographic findings in six patients with convulsive laughter, of whom four showed the 14 and 6 per second positive spike pattern and one showed a temporal focus. The electroencephalogram thus at times supported the possibility of the diencephalic origin of these seizures but at other times was suggestive of cortical or diffuse involvement.

The relation of epileptic laughter to the "forced" laughter seen in organic brain syndromes is perhaps worthy of speculation. This forced laughter is usually excessive, often without any associated affective component, and may be precipitated by inappropriate stimuli. Forced laughter has usually been explained as the result of reduced inhibition of a postulated center for laughter. Some of these patients demonstrated convulsive laughter at times and at other times had episodes of silly giggling without obviously epileptic sign, as for example case 10. It is difficult, if not impossible, to separate sharply convulsive laughter from forced laughter, for these conditions may indicate merely the relative importance of intrinsic activation and of reduced inhibition of a center for laughter.

Laughter also has some other neurologic correlates. Paskind[17] has reported a decrease in tone in normal individuals during laughing. Sudden decrease in tone may also occur following laughter in the condition known as cataplexy. The head-nodding attacks in case 9 may represent a further association of loss of tone with laughter.

The clinical picture in these patients with convulsive laughter has been quite varied. The history and physical examination and the electroencephalogram all show wide variations. They do have one factor in common, namely that in all of these patients the epilepsy was thought to have a symptomatic rather than an idiopathic or genetic basis.

The 11 cases reported herein would indicate that laughter as an epileptiform manifestation is more frequent than would be suspected from the few reports of this phenomenon in the literature.

SUMMARY

Eleven patients with laughter as an epileptiform manifestation have been described. The laughter varied from violent prolonged laughter lasting two minutes or so to giggling and even grinning. The epileptic origin of the laughter was indicated by the lack of any external precipitant, by the nature of the laughter, by concomitant manifestations of epilepsy, and by response to anticonvulsant medication. The clinical and the electroencephalographic picture of these patients showed much variation, but in general indicated the probability of an organic lesion of the brain as the causative factor. The possible role of the hypothalamus in the production of this seizure manifestation has been discussed.

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■ To try to solve the very important but complicated questions which are so numerous in both the physiological and the pathological history of the parts of these nerve-fibers which are in the nervous centres, it is necessary to make use of all the means that science may furnish, and particularly of the two best, which are: experimentation upon living animals, and observation of pathological cases. When employed together, these means of scientific research have allowed errors to be made; but, of course, the chance of committing errors is by far greater when either of them is employed alone. The danger of making use of one of these means exclusively is very strikingly illustrated by the many errors, concerning the cerebellum, committed by experimental physiologists, who mistook the effects of certain circumstances of their experiments for the results of injuries or of the absence of the cerebellum. Had they taken the trouble of comparing the phenomena they saw with those observed by medical men in cases of disease of the cerebellum, they would not have introduced in science a number of hypotheses which impede its progress. It is by so doing that experimentalists have thrown discredit on the means of scientific inquiry of which they have made so much use and abuse, and have given ground to many critics to blame their means of research, while the fault was in the men who employed those means, and not in the means themselves.

C. E. Brown-Séquard in *Lectures on the Physiology and Pathology of the Central Nervous System*, published in 1860.

Palatal Myoclonus

A New Approach to the Understanding of Its Production

Christian Herrmann, Jr., M.D., Paul H. Crandall, M.D. and Harry C. H. Fang, M.D.

PALATAL MYOCLONUS is an involuntary repetitive contraction of the soft palatal musculature at rates between 60 and 180 per minute. The movements are not always limited to the soft palate, but also frequently involve the pharynx, larynx, eyes, diaphragm, and musculature at the mouth of the eustachian tube. The latter may provoke a clicking sound in the ear or throat which is sometimes called eustachian tinnitus. In some cases the orbicularis oculi, facial, tongue, and limb muscles are also involved. The contractions of the various muscles and movements of the involved parts are generally synchronous at all points, even though geographically widely separated in the body.

Etiology of the disorder is unknown. It has been associated with a variety of underlying neuropathologic conditions. These include vascular infarctions involving brainstem, multiple sclerosis, aneurysm of the vertebral artery, tumor involving the brainstem or cerebellum, trauma, syphilis, malaria, and other more obscure degenerative processes. It has also been reported in association with heredofamilial tremor.

Palatal myocolnus is generally considered a rare disorder. However, eight cases have come to our attention within 19 months. This apparent high incidence and the variable associated clinical and pathologic findings prompted re-evaluation of the pathogenesis of this intriguing and infrequent phenomenon, since it does not conform to the classic characteristics of involuntary movements commonly seen in extrapyramidal motor disorders.

Five patients were observed in a Veterans Administration hospital, two on the neurologic and three on the neurosurgical wards. Three cases were seen in the university medical center. Cinematographic studies of the palatal myoclonus and associated motor phenomena were obtained in six cases.

CASE REPORTS

Case 1. A 55 year old married, white male was irritable, tired, nervous, and anorexic for three months preceding sudden collapse and coma on December 3, 1955. Lumbar puncture

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at another hospital was reported as grossly bloody. Tracheotomy was performed for adequate airway and control of secretions. He remained comatose for one month and was transferred to a second hospital, where a subsequent lumbar puncture on January 18, 1956 revealed a pressure of 100 mm., clear, colorless fluid, one lymphocyte per cu.mm., a negative Pandy, protein 32 mg. per cent, sugar 108 mg. per cent, and negative culture.

He was transferred to the Veterans Administration hospital and admitted to the neurologic service on January 27, 1956. He was a debilitated, middle-aged male responding only irregularly to simple commands; pulse 84; blood pressure 110/70; temperature 99.6° F. He made no attempt to speak. He had a tracheotomy tube and indwelling catheter. Left trochanteric and sacral decubitus ulcers were present. The carotid pulses were present and equal in the neck. The right pupil was fixed and dilated; the left was small and also fixed to light. The right cornea was ulcerated. The optic disks were normal. Eye movements on the right were limited except for conjugate horizontal gaze. There was a right facial paralysis of lower motor neuron type. There were continuous repetitive contractions of the soft palate, pharynx, and larynx at approximately 150 per minute. He was unable to swallow. Gag and palatal reflexes were feeble bilaterally. There was a left flaccid hemiplegia. Only feeble withdrawal responses to painful stimuli of the right arm could be elicited; there were none on the left. The deep tendon reflexes were brisk on the right, with slight increased resistance to passive movement on that side. Only feeble deep tendon reflexes were obtainable on the left, with equivocal plantar responses on that side.

Laboratory studies revealed a white count of 17,200 and hemoglobin of 11.6 gm. The serology was negative. Urine contained 2 plus protein, with many red and white blood cells. The blood urea nitrogen was 10 mg. per cent. The blood sugar was 235 mg. per cent. Serum electrolytes were normal. Chest roentgenogram showed elevation of the right diaphragm. Skull films were normal.

He received supportive therapy, including nasogastric tube feedings, insulin for his diabetes, and antibiotics to control the urinary tract infection and decubitus ulcers. The right eyelids were sutured partially shut to protect the cornea from further damage.

Palatal myoclonus has been continuous for four months since admission. He remains inconstantly responsive to simple commands.

Comment. In this patient palatal myoclonus appeared in association with moderately severe cerebral vascular insufficiency involving the brainstem in a middle-aged diabetic male. This most probably represents a thrombosis of a branch of the basilar artery. However, a ruptured aneurysm in the basi-vertebral system cannot be entirely excluded. Unfortunately, no exact time relationship between the cerebrovascular accident and the onset of the palatal myoclonus could be determined. From the clinical findings it would seem apparent that one or more lesions are present in the brainstem in the region of the pons and medulla. Midbrain involvement cannot be excluded in view of his lack of alertness and impaired responsiveness.

Case 2. A 55 year old separated, white lawyer had a myocardial infarct in 1949. In April 1954 he had polyuria, polydypsia, polyphagia, weight loss, and fatigability. Diabetes mellitus was discovered and treated by diet. In June 1954 he had a conjunctivitis and glaucoma with frontal headaches which subsided with treatment. On July 1 and 3 he had transient episodes of dizziness. On July 5 he suddenly collapsed without loss of consciousness while sitting at home. He had marked weakness of the right extremities, ataxia of the left extremities, dysarthric speech, and pain behind the left eye. He was admitted to another hospital where a lumbar puncture revealed a pressure of 140 mm., 1 white cell, and 41 mg. per cent protein. He remained there two months and was transferred to a rest home for another two months. He was started on insulin. In September 1954 he had onset of palatal myoclonus. In October he developed almost continuous jerking movements of the left forearm, along with constricting band-like headaches. A sister was said to have multiple sclerosis for 13 years.

On admission to the Veterans Hospital January 14, 1955, his blood pressure was 140/80. He had vertical nystagmus on upward gaze and a left Horner's syndrome. The right half of the face was hypesthetic and hypalgesic. Corneal reflexes were sluggish bilaterally. There was a left facial weakness. Rhythmic synchronous contractions of the soft palate, pharynx,

larynx, and facial muscles were noted on the left about the corner of the mouth extending into the platysma. The frequency varied between 120 to 130 per minute. A synchronous clicking sound was audible with a stethoscope over the bony surfaces of the cheeks, as well as over the open mouth — eustachian tinnitus of Bjork. He spoke with a markedly slurred dysarthric bursting speech.

There was trunkal titubation. He was unable to stand or walk without support. There was marked cerebellar incoordination and hypotonia of the limbs on the left and to a slight degree on the right. There was a slight deep reflex preponderance on the right, with an extensor plantar response on that side. An inconstant resting tremor of the left forearm occurred synchronously with the soft palate. The movements persisted during sleep. They were briefly interrupted with volitional effort such as speaking, holding the breath, and squeezing the hands. Pain, light touch, and temperature perception were lost over the right half of the trunk and extremities.

Laboratory studies revealed a normal hemogram and a negative urinalysis. Fasting blood sugar was 138 mg. per cent. Kolmer reaction was negative. Spinal fluid had a pressure of 140 mm. with no cells. Roentgenogram showed 35 mg. per cent protein, 62 mg. per cent sugar, and colloidal gold 000110000. Chest roentgenogram showed cardiomegaly. Chest fluoroscopy showed no apparent involuntary movements of the diaphragm.

He received insulin, physiotherapy, and a course of Dicumarol. There was only slight decrease in incoordination when he was discharged home on May 6, 1955. Re-examination two months later showed no change.

Comment. This middle-aged male with cardiovascular disease and diabetes mellitus developed palatal myoclonus following vascular insufficiency of the brainstem, most probably representing thrombosis in one or more of the intermediate and lateral vessels of the basivertebral system on the left, provoking infarction in these regions.

Case 3. A 38 year old married, Spanish-American male came to the neurology outpatient clinic on August 23, 1954, complaining of slowly progressive awkwardness of his hands and unsteadiness on his feet for the past ten years. He had slight blurred vision for nine years and occasional momentary diplopia on looking to either side or ahead. For three years his speech was slowed and slightly thick. His symptoms were worse with excitement. There were no remissions or exacerbations. He had fallen from a horse at age 12 and was unconscious approximately one hour. He consumed one quart of wine daily until 1950, but was supported by his family in denying frank drunkeness.

The right testis was undescended. Visual acuity was 10/20 minus 2 in OD, and 10/30 in OS. The optic disks had normal color. Slight continuous, spontaneous rhythmic, vertical nystagmus was present in both eyes in the primary position of forward gaze. Horizontal nystagmus with occasional dissociation of conjugate gaze was present on looking to either side. This was greater to the left and associated with a rotary component at times. Rotary nystagmus was increased on oblique upward gaze. The vertical nystagmus was increased on direct upward gaze. Lateral gaze to either side was slightly limited. Brief diplopia resulted on lateral gaze to either side, as well as on downward gaze. The pupils reacted sluggishly to light and accommodation. The corneal reflexes were sluggish. The jaw movements were normal. Continuous rhythmic contractions of the soft palate and posterior pharyngeal wall occurred at a rate of 140 per minute synchronous with the vertical nystagmus but unrelated to the pulse. Speech was low in pitch and volume, tremulous and slightly slowed in rate. His gait was unsteady and slightly reeling. He swayed in the Romberg position and could not stand on either foot alone with eyes opened or closed. Nonequilibratory coordination tests were performed, with unsteadiness in both the upper and lower extremities, most marked on the left. Succession movements were slowed bilaterally, but particularly on the left, in both upper and lower extremities. Check was not affected and rebound phenomenon was absent. The right knee jerk was slightly more active than the left. Plantar flexion was present on the left. No response was obtained on the right. Sensory and mental examinations were normal.

Skull films showed long, jointed styloid processes, but were otherwise normal. An electroencephalogram obtained elsewhere in July 1953 was said to show a spike-and-wave focus in the right occipital region at the rate of one to three per second, with amplitude of 30 to 40

microvolts. An electroencephalogram in our laboratory in March 1955 was irregular and mildly disorganized, with frequent five to seven per second and occasional four per second activity intermixed with eight to nine per second activity. The slow activity was most prominent in the frontal, temporal, and parietal areas. The record was considered diffusely, mildly abnormal without specific features. Lumbar puncture in August 1953 revealed a pressure of 80 mm. with three cells per cu.mm., a negative Kolmer reaction, and colloidal gold of 0011100000. Fluoroscopy in the fall of 1955 revealed slight rhythmic movements of the larynx at the same rate as those of the soft palate, using a swallow of thick barium paste. No abnormal movement of the diaphragm was noted.

He has shown no significant change in his symptoms or signs when re-examined at intervals during the past 18 months. He continues to work regularly in the orchards.

Comment. This case illustrates the occurrence of palatal myoclonus in association with an insidiously progressive cerebellar syndrome of probable degenerative nature. The possibility of multiple sclerosis might be considered, but onset and course have been insidious over ten years with no clear remissions or exacerbations. The signs are those of cerebellar and intra-brainstem dysfunction at the level of cerebellar connections. The role of his earlier alcoholic ingestion remains speculative.

Case 4. A 55 year old single white male was first admitted to the medical service of the University Hospital on November 16, 1955, with a history of hypertension and retinal hemorrhages allegedly for the preceding ten years. He had progressive visual loss and difficulty controlling his bowel and bladder for two years. He had numbness of the feet for three years and of the little fingers for one year. His gait had become increasingly unsteady for several months, requiring support to walk.

Examination on November 28, 1955 revealed a middle-aged male confined to bed with blood pressure of 205/85. He had an indwelling catheter and made restless movements of the lower extremities. His attention span was short. He was incompletely oriented for time.

He counted fingers at one and a half feet in OD and two feet in OS. The fundi had extensive old and recent hemorrhages and exudates. The right optic disk was pale, and the vasculature was obliterated locally. Pupillary reactions were sluggish. Upward gaze and convergence were limited. No abnormal involuntary movements of the soft palate or associated structures were noted. Snout reflex was present. He was right-handed and exhibited slight weakness in the small muscles of the hands. There was mild to moderate weakness in the lower extremities, greatest distally. Point-to-point and other coordination tests were poorly performed, especially in the lower extremities. Only feeble tendon reflexes were obtained in the upper extremities. They were absent in the lowers. The right plantar response was flexor and the left biphasic.

Touch perception was generally reduced in the fingers distally and in the feet up to the lower one-third of the legs. Vibratory perception was lost in the lower extremities and trunk up to the lower one-third of the rib margin anteriorly and to T-12 posteriorly. Deep pain was reduced in the heel tendons. Position sense was impaired in the toes. There was no nerve trunk tenderness. Laboratory studies revealed a blood urea nitrogen of 55 mg. per cent, hemoglobin 7.7 gm., and white count of 10,700. His urine contained 3+ protein, as well as cylindruria. Cerebrospinal fluid showed 140 mm. pressure, no cells, negative Kolmer reaction, negative colloidal gold, 63 mg. per cent protein, and 79 mg. per cent sugar. Muscle and skin biopsies were normal. There was roentgenographic evidence of cardiac enlargement and right pleural effusion. There was electrocardiographic evidence of digitalis effect and/or left ventricular strain. Cystometric studies showed a hypotonic bladder.

Following appropriate supportive medical therapy, he was dismissed home on December 13, 1955, with little change until December 20 when he had transient right facial weakness and difficulty speaking. This cleared in four to five hours but returned the day before his second admission on December 22. At that time it was more persistent, accompanied by inability to swallow and tendency to cough and choke. Weakness of the right extremities was not immediately apparent. He was lethargic but oriented. His speech was feeble, scant, and almost unintelligible. There was a right supranuclear facial weakness, gag reflexes were intact, and no involuntary movements of the palate were noted on December 22, 1955. Other findings were essentially the same as

before. He developed inspiratory rales and two plus pitting edema of the lower extremities. Lumbar puncture on December 22 revealed an initial pressure of 180 mm., two white cells, 46 mg. per cent sugar, 48 mg. per cent protein, and negative Kolmer reaction.

The following day he became somnolent. The right facial weakness increased. Blood urea nitrogen increased to 79 mg. per cent. Serum carbon dioxide fell to 11 millimoles per liter, the chlorides to 90 milliequivalents, sodium to 118.5 mEq., and potassium to 5.1 mEq. An electrocardiogram showed first and second degree A-V block and other changes consistent with myocardial ischemia and/or digitalis effect. A second neurologic consultation on December 30 revealed a stuporous male with deep respirations at 12. He responded very feebly to loud simple commands but did not move the tongue or lower limbs. The left pupil was smaller than the right and both reacted poorly. There were slow, roving movements of the eyes. The right corneal reflex was diminished. He yawned frequently, at which time the tongue became firm and the uvula elevated centrally. There were rhythmic contractions of the soft palate at approximately 156 per minute bilaterally. Gag reflex was present. He moved the right arm only feebly in response to painful stimuli and made occasional restless exploratory movements with the left. He moved the lower extremities only on painful stimuli. The left moved more promptly than the right. Deep tendon reflexes were feeble to absent bilaterally. The legs and abdomen were edematous. A feeble right Babinski was present, as well as a snout reflex. He remained poorly responsive and his urinary output declined, despite adequate fluids. On January 2, 1956 he vomited coffee grounds material and became pale and cyanotic. His respirations were Cheyne-Stokes briefly, followed by absent pulse, blood pressure, and heart sounds, with ultimate cessation of respiration. Autopsy was refused.

Comment. This patient with hypertensive cardiovascular renal disease and renal failure also had diffuse vascular involvement of the brain, including cerebrum and brainstem, accompanied by palatal myoclonus. In addition, there were features of a peripheral neuropathy.

Case 5. A 75 year old married, retired physician was seen on November 30, 1955 as an outpatient, complainng of tremor of the hands increasing the previous three years. It occurred chiefly when using the hands and was worse when excited. At these times the fingers and palms tended to flop without significant movement of the proximal upper limbs. Movements were worse on the right and, when the patient was tense, appeared on that side at rest. There was little change in the character of the movements the preceding year. He had had moderate hypertension for several years. A maternal uncle and aunt died at about 80 with antecedent tremor of the hands similar to that of the patient for some ten years. The patient's brother, age 90, had a 20-year history of tremors of the hands which had not changed in intensity for several years. Five other siblings were dead without history of tremor. There was no history of mental disorder or loss of memory in the family known to the patient.

The patient was alert and responded quickly for his age. Blood pressure was 170/105. There were no cardiac abnormalities. The extraocular movements were normal except for slight unsteadiness when gazing quickly from one side to the other. There was no nystagmus. Occasional slight rhythmic contractions of the eyelids occurred. These were synchronous with rhythmic contractions of the soft palate. The latter were not synchronous with the pulse. The tongue was quiet in the floor of the mouth, but when protruded it exhibited a coarse to-and-fro tremor along with the jaw. These movements were not synchronous with those of the palate.

Coarse, somewhat flopping intention tremor of the fingers and wrists occurred with voluntary effort, worse on the right and terminally in the course of movement. Skilled acts such as writing were almost impossible to perform. Occasionally there was tremor at rest in the right hand. This was not synchronous with the palatal movements. Resistance to passive movement was not increased in the extremities. The deep reflexes were active and equal except for sluggish ankle jerks. Plantar responses were flexor. Gait and coordination were normal in the feet. Sensation was intact except for impaired vibration in the knees and ankles. The Romberg sign was negative. He was given reserpine 0.25 mg. t.i.d., with partial relief of the intention tremor but no change in the palatal myoclonus.

Comment. A patient with familial tremor

illustrates its occasional association with palatal myoclonus.

Case 6. A 58 year old male first noted diplopia and blurred vision in 1934. In 1938 he had staggering gait and dragged his right foot. In 1937 he had a tremor in the right upper extremity. Examination showed lateral and rotary nystagmus, intention tremor of the right upper extremity, and ataxic, scuffing gait on the right, with inability to stand on that foot alone. His symptoms and signs remained relatively stationary until 1948 when he complained of headache when lying on the right side. Re-examination showed impairment of alternating movements and past-pointing in the right upper extremity. Position sense was found to be somewhat impaired in the right hand. He was treated for a basal cell carcinoma below the left eye.

His symptoms and signs increased slowly. He was readmitted to the Veterans Hospital in 1950, complaining of headache on exposure to the sun and on certain postural changes. He had a numb feeling in the right side of his face, and hearing in the right ear was intermittently impaired. He then had nystagmus, slight drooping of the right eyelid, lagging of the extraocular movements on the right, diminished right corneal reflex, flattened right nasolabial fold, and diminished gag reflex. He had dysdiadokokinesis and past-pointing on the right and very unsteady gait, with a positive Romberg sign. Spinal fluid examination in 1950 revealed a protein of 92.5 mg. per cent; in 1952 it was 103 mg. per cent.

In January 1953 the patient had increased difficulty in swallowing liquids. With coughing he experienced sharp pain in the right side of the face. He continued to have intermittent severe throbbing headaches which became continuous for ten days in October 1953, accompanied by sleepiness and drowsiness. He had been unable to walk since February 1953 and subsequently developed nocturnal enuresis and slurred speech.

Re-examination revealed bilateral papilledema with extensive hemorrhages about the disks. Acuity was reduced to large block letters. His facies was expressionless. Fasciculations were seen in the tongue. The deep reflexes were more active on the left, and a left Babinski sign was present. Skull films showed enlargement of the right internal auditory meatus. Suboccipital craniotomy on October 19, 1953 disclosed a glistening, pearly white lobulated mass, extending from the second cervical vertebra upward to the right cerebellopontine angle and encroaching on the right cerebellar hemisphere so that the fourth ventricle was deviated and ballooned out. During removal of the tumor one major branch of the right posterior inferior cerebellar artery was clipped. Pathologic examination revealed the tumor to be epidermoid (figure 1 A and B). He had a slow postoperative recovery, with marked tremulous movements of all limbs decreasing over the next month. He also had a

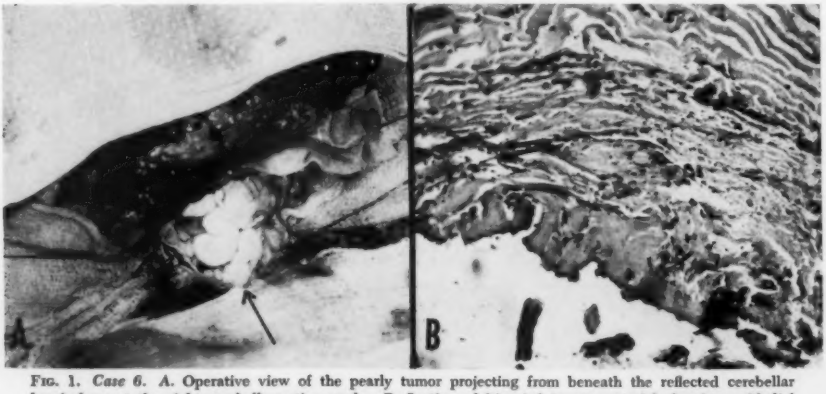

FIG. 1. *Case 6. A.* Operative view of the pearly tumor projecting from beneath the reflected cerebellar hemisphere at the right cerebellopontine angle. *B.* Section of biopsied tumor material showing epithelial stratification and lamellation of collagen and focal deposits of malignant cells. Iron hematoxylin-eosin stain.

right abducens palsy and internal strabismus on that side.

In 1954 the patient and his wife noted continuous clicking noises in his throat but could not discover the cause. He was readmitted to the Veterans Hospital in August 1955 for two basal cell carcinomata of the face. On examination he was obese and confined to a wheel chair because of a severe ataxic gait. There was an internal strabismus, with inward deviation of the right eye and right abducens palsy on lateral gaze to the right. The right pupil was slightly larger than the left. There was slight weakness of the obicularis oculi and widened palpebral fissure on the right. He had nystagmus of fixation type in all directions of gaze. The right corneal reflex was diminished. There were continuous rhythmic myoclonic contractions of the soft palate and larynx which were briefly halted by voluntary elevation of the palate. There was a tracheal tug synchronous with the palatal myoclonus. Intermittent myoclonic movements were noted in the right frontalis, mentalis, and alae nasi muscles. The movements were not synchronous with the pulse. His voice was extremely dysarthric, with a rather low growling quality and intermittent interruptions in the production of sound. There were marked right cerebellar signs, including terminal intention tremor, past-pointing, rebound phenomenon, dysdiadokinesis, and markedly impaired right knee-shin test. When the patient held his hand outstretched, there were intermittent myoclonic movements of the index and ring fingers which did not occur at rest or with extreme effort. He had a soft pulsating right suboccipital cranial defect.

Electromyographic studies showed rhythmic discharges of normal motor units in the muscles of the soft palate. These were synchronous with the myoclonus and suggestive of an upper motor neuron lesion. Examination of the muscles of the face and neck where the myoclonus was observed clinically showed no electrical discharges. Fluoroscopic examination showed no involvement of the diaphragm, but a swallow of thick barium paste revealed myoclonus involving the pharyngeal and laryngeal musculature. The right vocal cord moved slower than the left. An audiogram showed mild perceptive deafness in the right ear, but the left gave normal responses. The patient was seen for a follow-up visit early in 1956 and continued to have the same movements and findings as noted earlier.

Comment. This patient had a slow-growing epidermoid extending from the right cerebellopontine angle down to the second cervical vertebra. He had this tumor removed. It was also necessary to clip a major branch of the posterior inferior cerebellar artery at the time of surgery. Palatal myoclonus developed one year later.

Case 7. A 56 year old white male had been followed at the Veterans Hospital since April 1950. For 15 years he complained of intermittent pain in the left mastoid region when turning his head suddenly to the left. For five years he had intermittent pain in the shoulder, excruciatingly severe in 1950, radiating from the base of the neck into both shoulders and anterior aspect of both arms. It did not go below the elbows. It was worse in the day and relieved at night. For one month he had had recurrent nausea, vomiting, headache, ataxic gait, anorexia, and fatigue. His family had noted intermittent episodes lasting for a few seconds, during which he was confused. Examination at that time revealed a bilateral papilledema. The right pupil was larger than the left and reacted poorly to light and convergence. There was no nystagmus. There was tenderness on light palpation of the left mastoid area. He had a wide-based staggering, ataxic gait with tendency to fall to the right. There was intention tremor and rebound phenomenon in the left upper extremity. Spinal fluid protein was 62 mg. per cent. Serology was negative. Ventriculogram on May 1, 1950 showed moderate dilation of the lateral and third ventricles and no filling of the fourth ventricle. Suboccipital craniectomy revealed thickened arachnoid over the fourth ventricle, but no tumor was discovered. He had a slow postoperative recovery and remained ataxic.

In April 1953 he was assaulted, beaten, and apparently sustained a contusion. He continued to complain of recurrent posterior occipital headache, neck pain, visual blurring, increased ataxia, and disequilibrium. On examination at that time his memory was impaired and he frequently confabulated. He had mild papilledema, with obliterated disk margins and flame-shaped hemorrhage in each fundus. Left facial weakness was noted. His gait was broad-based and staggering. He seemed to have more trunkal and lower extremity ataxia than in the upper extremities. A second suboccipital cra-

niotomy on April 14, 1953 disclosed necrotic transverse foliae in the vermis. A mass the size of a large English walnut was encountered at 3 to 4 mm. depth and removed. It was soft, vascular, and contained a cyst filled with thick yellow fluid. It lay over the roof of the fourth ventricle and occluded the aqueduct. It was attached to the choroid plexus of the fourth ventricle. Pathologic examination of the tissue was reported as hemangioendothelioma. Postoperatively his speech was slurred to the point of being incoherent. Swallowing was difficult and he complained of constant lumbar backache.

In 1954 he continued to have difficulty speaking and initiating deglutition so that saliva drooled from his mouth. He also complained of spells of vertigo, weakness in both legs, and pain in the low back and in both legs. He was admitted again in January 1955. He was a thin, elderly white male, unable to walk because of ataxia and standing only with support. When sitting he had a constant, coarse, slow tremor involving the jaw, trunk, neck muscles, and proximal limb muscles. This was exaggerated by any activity and was slightly more marked on the left. It occurred at a rate of about two or three per second. In addition, there was bilateral rhythmic palatal myoclonus, synchronous with the tremors of the extremities. There was no nystagmus. If the patient had been active, the rhythmic tremors also appeared to involve the fingers and frontalis muscles. He had marked dysarthria and his voice fluctuated in volume. He could not perform fine finger movements or alternating movements. There was bilateral past-pointing and decomposition of movement. The pupils reacted sluggishly to light, and there was a chronic low-grade papilledema with patches of exudate in the left fundus. The visual acuity was Jaeger 14 left and Jaeger 18 right. Position sense was impaired in the lower extremities, more on the left than on the right. Fasciculations were observed in the lower extremities. Straight leg raising was limited to 20 degrees in both lower extremities. The left ankle jerk was absent. A left Babinski sign was present.

On January 21, 1955 percutaneous vertebral arteriography showed a huge vascular tumor in the midline of the cerebellum, extending from the tentorium to below the foramen magnum (figure 2A). A third suboccipital craniotomy was performed on January 24, 1955. The tumor mass completely replaced the vermis and three-fourths of each cerebellar hemisphere. It was mobilized from the floor of the fourth ventricle up to the midbrain. Pathologic examination showed a 60 gm. irregular globular mass composed of numerous blood vessels, large vascular spaces, and delicate fibrous stoma with large areas of soft, dull grey homogeneous material (figure 2B, C, E, and F). Histologic diagnosis was hemangioblastoma. A left ureterosubarachnoid anastomosis was complicated by pneumonia and meningitis from which the patient failed to recover.

Comment. This patient with long-standing cerebellar symptoms and signs associated with an expanding midline intracerebellar hemangioblastoma was observed to have palatal myoclonus approximately one year following subtotal removal of the tumor.

Autopsy report. At necropsy the general pathologic findings included status post lumbar subarachnoid ureterostomy and left nephrectomy; acute pleuritis, left lung; bronchopneumonia, right lung, with abscess formation; acute duodenal ulcers; cholelithiasis; and emphysema and interstitial fibrosis, left lung.

Neuropathologic findings. The brain weighed 1,500 gm. The leptomeninges in the basal portions of the brain were brown and thickened. Almost all of the central two-thirds of the cerebellum was absent. The crater thus formed was lined by hemorrhagic soft material. This extended to the roof of the fourth ventricle. The entire ventricular system was moderately dilated. The ventricles were lined by a dull grey-brown ependyma. Dull grey fibrinous material was present over the entire arachnoid of the spinal cord. There was a complete occlusion of the lumen of the basilar artery (figure 2D).

Microscopic findings. Representative sections of the operative areas showed hemorrhage, presence of gitter cells, foreign body giant cells, polymorphonuclear leukocytes, lymphocytes, and gliosis along the margins of resections. This extended further into the fourth ventricle. Cerebrum showed moderate edema and slight satellitosis of the nerve cells. There was a diffuse acute ependymitis, acute and subacute meningitis. Inflammatory cells were found to cuff around the perivascular spaces of the brain and spinal cord. The basilar artery showed acute arteritis.

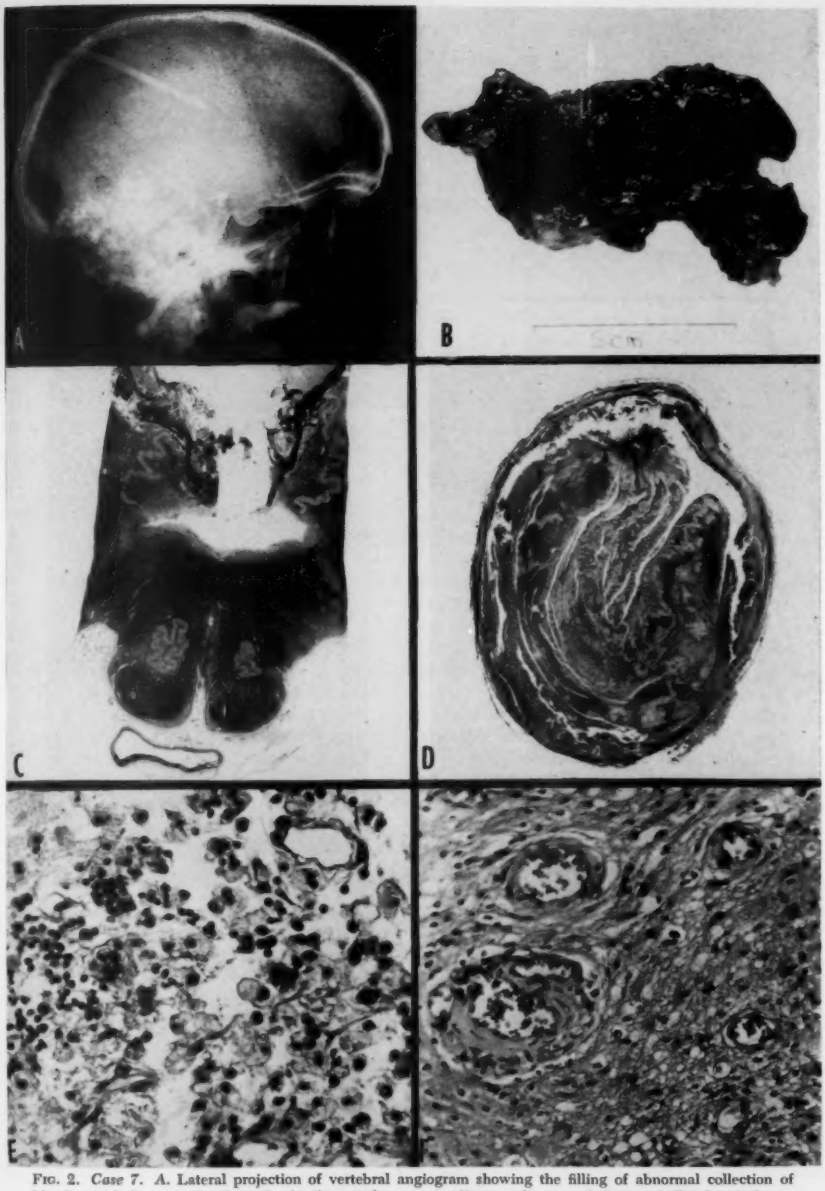

FIG. 2. *Case 7. A.* Lateral projection of vertebral angiogram showing the filling of abnormal collection of blood vessels located dorsal to the basilar trunk corresponding to the site of the cerebellar hemispheres. *B.* Gross appearance of the resected tumor mass from cerebellum. *C.* Horizontal section of upper medulla and cerebellum showing hemangioblastoma replacing most of the vermis. It extended to the roof of the fourth ventricle. Both dentate nuclei were compressed laterally. Weil stain for myelin sheath. *D.* Section of middle third of the basilar artery showing acute arteritis and laminated thrombus occluding the lumen. Iron hematoxylin-eosin stain. *E.* Section of resected tumor tissue from cerebellum (with high magnification) showing typical collection of foam cells as well as extravasated hematogenous elements. Iron hematoxylin-eosin stain. *F.* Section of tumor demonstrating the hyperplasia of abnormal blood vessels and fenestrated appearance of the stroma, due, in part, to the foam cells. Iron hematoxylin-eosin stain.

Case 8. A 36 year old Mexican male bricklayer complained of frequent dull headaches and occasional diplopia for five years. In 1949 he was rendered unconscious in an auto accident. This was followed by several months of persistent headache. In 1953 he had rather abrupt loss of visual acuity in the left eye. After a few days he had only light perception. This was associated with scintillating scotoma and periorbital pain. These lasted four weeks and then disappeared completely. At that time an ophthalmologist made a diagnosis of optic neuritis.

About October 1, 1954 the patient had abrupt onset of suboccipital headaches, vertigo, difficulty speaking, diplopia, loss of memory for recent events, and numbness of the right arm and leg. He became ataxic and a few days later was unable to walk. He had nausea and severe tinnitus on the left.

He was admitted to the Veterans Administration Hospital on October 20, 1954. He had a slight right hemiparesis, with a right supranuclear facial weakness, flexion deformity of the upper extremity, and stiffness of the lower extremity on that side. He had a hemihypalgesia on the right. There was also slight impairment of light touch and position sense on the right.

Repeated lumbar punctures revealed pressure at the upper limits of normal and a pleocytosis between 60 to 220 white cells, the majority being lymphocytes. The protein ranged between 104 and 264 mg. per cent, with a 3+ to 4+ globulin. The serology was negative. Cerebrospinal fluid sugar was 21 mg. per cent. One electroencephalogram showed a left transient slow-wave focus. Two subsequent electroencephalograms awake and asleep in November 1954 were normal.

On December 2, 1954 a ventriculogram revealed an obstructive hydrocephalus with dilated lateral and third ventricles. No air passed beyond the posterior one-third of the third ventricle (figure 3A). On December 7, 1954 a suboccipital craniectomy showed no abnormality of the lateral recesses or fourth ventricle except for flecks of fibrin in the cerebrospinal fluid. The ventricular fluid contained 62 lymphocytes, 104 mg. per cent of protein, and 4+ globulin. A Torkildsen procedure was then performed.

Following this the patient improved, showing only slight weakness of the left palate and slight tremor of the outstretched hands. He had mild stiffness of the neck, but worked regularly until July 1955 when he had a recurrence of all the earlier symptoms except that the numbness only returned to the distal portion of the right arm and he developed a left-sided Horner's syndrome.

He was readmitted to the Veterans Administration Hospital in August 1955. His condition deteriorated. He developed a reeling gait and changes in the extraocular movements which were difficult to clarify. There appeared to be impairment of conjugate supra- and infravergence. The left pupil reacted poorly to light and was smaller than the right. Lumbar puncture revealed a normal pressure. There were 82 white cells with 80 lymphocytes. Protein was 340 mg. per cent, with 4+ globulin and 58 mg. per cent sugar. Dye tests demonstrated that the Torkildsen tube was functioning.

A pneumoencephalogram showed the fourth ventricle patent up to the posterior limit of the cerebral aqueduct (figure 3B). On August 24 a vertebral arteriogram revealed that the basilar artery was smoothly outlined but considerably compressed and displaced against the clivus. Only a small amount of dye passed into the posterior cerebral arteries. When 15 cc. of Diodrast was used, there was a reflux of dye through the subclavian artery into the internal carotid and anterior and middle cerebral arteries at the same time as the demonstration of the vertebral system (figure 3C). Numerous cultures and preparations for acid-fast bacilli, fungi, torula, and parasites were made on the cerebrospinal fluid. All were negative. The patient had a 2+ skin reaction to coccidioidin one to one thousand, but the complement fixation test for this was negative. Tests for blastomycin and histoplasmin were negative. Blood and spinal fluid serologic reactions were negative. The patient was discharged from the hospital without significant improvement.

In March 1956 he developed nocturnal chills, difficulty with urination, and intermittent burning sensation of the right face.

He was readmitted to the hospital. He was oriented, but had poor memory, digit retention, and reasoning power. His general background of information was very poor. The midline suboccipital scar was well healed and not tense or pulsating. His gait was slow, with the right upper extremity held flexed and slight extension of the fingers and the right lower extremity stiff without smooth action at the

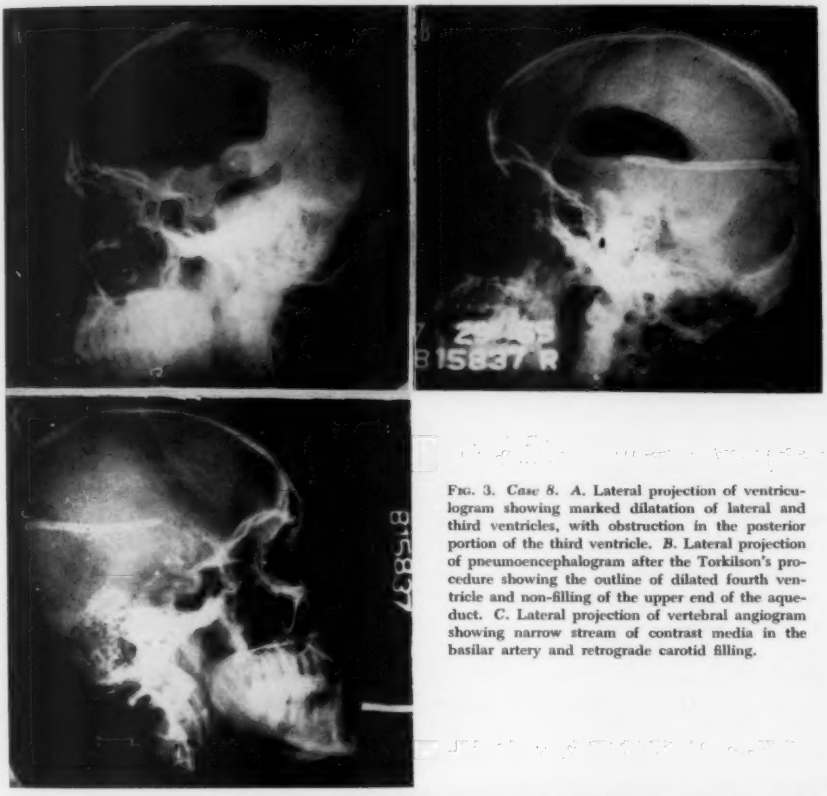

FIG. 3. *Case 8. A.* Lateral projection of ventriculogram showing marked dilatation of lateral and third ventricles, with obstruction in the posterior portion of the third ventricle. *B.* Lateral projection of pneumoencephalogram after the Torkilson's procedure showing the outline of dilated fourth ventricle and non-filling of the upper end of the aqueduct. *C.* Lateral projection of vertebral angiogram showing narrow stream of contrast media in the basilar artery and retrograde carotid filling.

knees. He complained of vertigo on arising and canted his head to the left. Peripheral visual fields were slightly constricted symmetrically. The right pupil was larger than the left. Left pupil reacted slowly and poorly to light and better to convergence. He had skew deviation on lateral upward gaze. Superior conjugate gaze was impaired, particularly in the right eye. He had a continuous rotary and vertical oscillation of the left eye which was synchronous with a left-sided unilateral palatal myoclonus at a frequency of 130 per minute. It was not altered by breath holding or carotid stimulation. It was interrupted only briefly by phonation.

His tongue deviated slightly to the right. He showed a slightly decreased strength in the right extremities. Only slow extensor and flexor movements of the fingers were possible on that side. He was unable to manage buttons or fine finger movements with the right hand. There was no change in resistance to passive movement. He had poor coordination in the right hand. No abnormal movements were seen in the extremities. Deep tendon reflexes on the right were more active than on the left. The right abdominal reflexes were only feebly present and distinctly less than on the left.

He continued to show a hemihypalgesia on the right, with a diminished corneal reflex on that side. There was also slight diminution of touch, vibration, and position sense on that side. The left Horner's syndrome with miosis and anhydrosis persisted.

Comment. This patient with unilateral palatal myoclonus on the left has remained a

diagnostic problem despite extensive studies. Certain of the evanescent symptoms and signs in his course could be explained on the basis of his basilar artery insufficiency, which is supported by the angiographic findings. It is difficult to explain the obstruction of the posterior portion of the third ventricle and upper end of the aqueduct on this basis. Neoplasm or granuloma of the upper pons and midbrain was strongly suspected but not confirmed at the time of suboccipital craniotomy. The elevated spinal fluid protein would be compatible with neoplasm or chronic inflammatory process. The latter is suggested by the elevated cell count in the spinal fluid, yet extensive studies to establish an infectious process and agent have proved negative.

Palatal myoclonus has been known for almost a century. Gallet[9] reviewed the literature on this disorder extensively in his thesis of 1927 and credits Politzer in 1862 with earliest description of the movements. Spencer[20] in 1886 was also among the first to publish a case report. Guillain and Mollaret[12] in 1931, Leshin and Stone[16] in 1931, Riley and Brock[19] in 1933, Bender and co-workers[1] in 1952, and Nathanson[18] in 1956 presented additional cases.

In view of the heterogeneous pathology associated with this phenomenon, it becomes apparent that palatal myoclonus is not a disease but a symptom and sign of disturbed function of the nervous system. In this respect, it is similar to the phenomenon of the convulsive disorder. Other associated manifestations of palatal myoclonus have been observed and reported by workers in allied fields, such as otolaryngology and ophthalmology. This may tend to fragment available figures of over-all incidence. They have placed emphasis upon an isolated sign of what is probably only a part of a more extensive clinical phenomenon.

The exact incidence of palatal myoclonus is not known. Although the present group of cases ranged between 36 and 75 years, other authors have noted the movement in children in their teens, as well as in octogenarians. It seems to be more common in middle and later life. The present group of eight cases were all white males. This is partially explained by the fact that five cases occurred among a Veterans Administration Hospital population which is almost exclusively male. Other cases in the literature have been approximately equally divided between male and female without particular predilection for any racial group. It is possible that palatal myoclonus occurs much more frequently than it is recognized. It may easily be overlooked during the routine physical and neurologic examinations unless one is particularly aware of the possibility of this phenomenon or the patient complains of clicking sounds in the throat or ear. As the average age of our population increases, the likelihood of cerebral vascular accidents also increases. This makes the likelihood of brainstem infarction greater, as well as the occasional associated complication of palatal myoclonus.

The exact time of onset of the involuntary movements in relation to cerebral vascular accidents of the brainstem, trauma, and expanding lesions of the posterior fossa is difficult to determine. In our case 4 they began within eight days of a vascular accident involving the brainstem. In case 2 they became apparent to the patient two months following brainstem infarction. In case 6 the patient became aware of a clicking noise in the throat nine or ten months following the removal of a left cerebellopontine angle epidermoid in which a major branch of the left posterior inferior cerebellar artery was clipped at

operation. In case 7 they were first noted 21 months following subtotal removal of a hemangioblastoma in the roof of the cerebellum. Whether the palatal movements were present before they were noted by the observer or the patient remains speculative. However, there does appear to be a variable delay between the vascular accident or operative trauma and the onset or recognition of the movements. The determining factors in this regard remain unclear.

The movements, once established, unlike other involuntary movements, tend to persist continuously throughout the remainder of the patient's life. They do not disappear in natural sleep, during phonation, or intravenous barbiturate-induced sleep. They have been observed to persist during carotid sinus stimulation, caloric stimulation, coma, hemiplegia, and impending death.

The exact pathophysiologic mechanism underlying the production of palatal myoclonus remains unknown. Guillain and Mollaret[12] believed that an interruption of neural conduction of the triangular pathways formed by the fiber connections between the homolateral dentate nucleus, contralateral red nucleus, and contralateral inferior olive was capable of provoking these movements. Freeman[8] concluded from his cases that a lesion of the central tegmental tract evoked the palatal myoclonus. Pseudohypertrophy of the inferior olive in which the normal cells of the olive are replaced by astrocytes has been considered another possible cause of the disorder. Nathanson,[18] in his most recent study of three autopsied cases, states that the primary pathology is in the homolateral dentate nucleus with secondary degenerative changes of pseudohypertrophy in the contralateral inferior olive. Review of pertinent literature fails to uncover a single case of isolated involvement of the red nucleus. The extent, composition, and function of the central tegmental tract remains poorly defined and inadequately understood.

On the basis of careful analysis of more than 20 autopsied cases[1,5–8,11–14,18,21] of palatal myoclonus up to March 1956, it is safe to conclude that no single lesion involving a single nervous pathway or nuclear link has been known to cause the appearance of these movements. On the other hand, there is abundant evidence favoring the view that multiple lesions of whatever nature in the brainstem in or near the olivodentate pathways give rise to this curious phenomenon of palatal myoclonus. Various investigators have called attention to one or another involved structure as the possible explanation for the myoclonus. The evidence for these pathogenetic claims is inconclusive to date.

Weinstein and Bender[23] succeeded in provoking myoclonic movements of the palate, tongue, lips, and associated areas when they stimulated the hilar region of the inferior olive in monkeys. Nathanson[18] reported preliminary additional studies by Bender and Teng showing that destruction in the region of one inferior olive of monkeys provoked typical continuous palatal myoclonus in those animals. These movements did not change with natural or barbiturate-induced sleep. Magoun and co-workers[17] have not noted this phenomenon with lesions in their animals elsewhere in the brainstem. They qualified this with the possibility that the experimental neurophysiologist is frequently observing other phenomena and is unaware of the clinical phenomena of palatal myoclonus or may not have kept his animal prepa-

ration a sufficient time for the involuntary movements to develop.

We submit that the phenomenon of palatal myoclonus may be considered a type of involuntary movement of extrapyramidal origin, using the term extrapyramidal in the broadest sense. It differs markedly from the other more common extrapyramidal involuntary movements such as parkinsonism, chorea, athetosis, dystonia, and ballism, as has already been mentioned. It is uninfluenced by spontaneous or drug-induced sleep, voluntary movement, coma, and impending death. It appears to be uninfluenced by the reticular system of the brainstem, in the light of our present knowledge regarding its activating and inhibiting mechanisms. Although the pathologic findings may involve only one dentate nucleus and a contralateral inferior olive, the clinical manifestations are most commonly bilateral. In view of the apparent delay between damage to the brainstem or cerebellum and development of the palatal movements, one may ask whether a period of adjustment takes place within the nervous system before the release of the abnormal movements. The muscles frequently involved do not represent any particular associated group from the evolutionary or comparative anatomic standpoint.[3] No pace-making center has been demonstrated clinically or experimentally for the production of these movements, but it seems likely that such a system or center may, in fact, exist. This would then be independent of the usual extrapyramidal system considered to exist at upper brainstem and higher regions and the reticular system of the brainstem as we currently understand them. This seems probable since the movements are independent of, different in distribution from, and persist under conditions in which other abnormal involuntary movements cease. Only basic vital functions such as respiration dependent on brainstem mechanisms persist. In this respect, it may be considered an extrapyramidal disorder of the lower level of the neuraxis, namely, the brainstem including cerebellum.

In view of these possibilities, additional study seems warranted to determine the nature and location of a pacemaker, if indeed one exists for this phenomenon. This may reveal hitherto unrecognized neural systems held in abeyance by higher centers or activated by imbalance provoked when portions of the normally functioning neural pathways are interrupted or destroyed.

SUMMARY AND CONCLUSIONS

1. The case histories and findings in eight patients with palatal myoclonus and associated conditions are presented.

2. The variety of factors limiting our knowledge concerning the clinical recognition and incidence of this phenomenon has been discussed.

3. The associated pathology was documented by therapeutic biopsy in one case and autopsy in another case.

4. The pertinent literature on clinical, pathologic, and pathophysiologic aspects of this phenomenon was reviewed.

5. It is suggested that palatal myoclonus may represent another type of extrapyramidal disorder arising from disturbed neural mechanisms in the brainstem including cerebellum.

The authors wish to acknowledge the interest and helpful guidance of Dr. August S. Rose, as well as his generosity for permission to include case 5 in this series.

We are also grateful to Mr. Philip P. Bleicher for his technical assistance in the cinematographic studies, and to Mr. William L. Martinsen for other illustrations.

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■ Galen describes very minutely, and, generally speaking, very accurately, the distribution of the nerves, from the brain and spinal marrow to the various parts of the body. He is of opinion that the mind acts upon the body by means of the animal spirit and nerves; and that the will being placed in the brain, at the origin of the nerves, gives the principle or beginning of motion to the first nerves, and by them to the muscles. He thinks that some of the nerves, but not all of them, are hollow tubes. Speaking of the optic nerves, he says that they have foramina; and that, in the dissection of large animals, a lucid spirit may be seen flowing through them both at their origin and at their insertion into the eyes. He admits the possibility that the spirit may flow through the minute branches of nerves, though, on account of their extreme tenuity, it is not visible; but he inclines to think that the nerves are not all hollow tubes, and that in some cases the nervous power is propagated by a kind of impulse given to the nerve.

John Cooke in *A Treatise on Nervous Diseases*, published in 1824.

Further Observations in Craniovascular Studies

Percy Brazil, M.D. and Arnold P. Friedman, M.D.

THE THEORY of vascular headache is predicated upon certain changes which occur in the external carotid circulation. These changes may involve alterations in the volume pulsations of part of the extracranial arterial system, as in migraine, or may involve alterations in the availability of oxygenated blood for the extracranial muscles, as in tension or muscular contraction headache.

In the past decade considerable attention has been focused on the problem of measuring the volume and pressure changes of the external carotid arteries.[1–4] Until recently, the only technics available for such study have been 1) intra-arterial puncture for the measurement of pressure changes, 2) use of glycerine pelottes for the measurement of volume changes, and 3) use of reflected or transilluminated light for the measurement of volume changes.[5]

Intra-arterial puncture of any of the branches of the external carotid artery is not a practical method for studying adequate numbers of patients. The glycerine pelotte and photoelectric technics are certainly more practical, but they are, unfortunately, endowed with too many variables.

The best available method yet devised for studying the external carotid circulation is the use of displacement transducers. These transducers are microphones with subaudible frequency responses. They are extremely responsive to small displacements and frequencies encountered in various physiologic activities. Pulsations of very small vessels can be picked up and recorded easily. The type of transducer used has a definite electrical capacity which corresponds to the pressure with which the transducer is applied to the blood vessel under study. Thus, by measuring the capacity of the transducer, which can easily be accomplished by incorporating a calibrator in the circuit, the pressure of application of the transducer can be recorded with considerable accuracy (within 3 mm. of Hg). Using this technic, it is possible to perform repeat craniovascular studies on patients with considerable validity, for the pick-up can be reapplied to an artery with a standardized pressure as often as desired. The only source of error which remains is the positioning of the pick-up with respect to the artery. This variable can be minimized by measuring fixed points of

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application on the skull from constant points of reference. The actual pulse wave obtained and recorded by a displacement transducer is similar to that obtained with the glycerine pelotte and photoelectric cell technics.

In the present study, we have been concerned with the effects of a variety of drugs on the pulse contour of the temporal, occipital, and supraorbital arteries.

The changes which are observed in the pulse wave after the use of a drug are essentially related to the size of the amplitude of pulsation and to the pulse rate. We have previously indicated that the initially observed size of the pulse wave can give no absolute information as to whether the vessel is dilated or constricted. It must be stressed further that a change in the pulse wave involving greater amplitude of pulsation does not necessarily imply dilation, nor does diminution in the pulse wave amplitude necessarily imply vasoconstriction.

In volumetric measurement of the pulses, the difference between the systolic and diastolic size of the artery is being recorded. Thus, in a constricted vessel, if the systolic force is very great, the pulse amplitude can be marked, giving rise to a relatively large pulsation; conversely, in a dilated vessel, if the systolic force is slight, the pulse amplitude will be small and give rise to a small pulsation. This is further complicated by the use of drugs which are called "dilators" and "constrictors." These are vague terms and should be clarified by indicating what it is that is being dilated or constricted. For example, nicotinic acid is loosely termed a dilator; yet it has been shown that it certainly does not dilate the intracerebral vessels and, in fact, produces the opposite effect.[6] Nicotinic acid, however, does dilate the vessels of the extracranial system. Thus, nicotinic acid is a constrictor (intracranial) and a dilator (extracranial) at the same time. This must be carried even further. When we say "extracranial dilator," do we mean that the large vessels (temporal, occipital, and so on) are dilated, or do we mean that the arterioles of these vessels are dilated, or do we mean that both are dilated? This is not a purely academic question, but is of direct importance when the pulse wave changes are interpreted. For if, after administering nicotinic acid, the arterioles are dilated proportionately more than the parent vessel, blood will be diverted faster and the pulse amplitude of the parent artery may not be increased even though there is general dilatation of the system. Or even if the vessel itself dilates, the systolic force may be lessened so that the pulse wave amplitude becomes diminished. This would explain why pulse wave amplitudes are often diminished after nicotinic acid is given. From the foregoing, the following observations are suggested: 1) The initial size of the pulse wave amplitude does not give absolute information about the resting state of the vessel. 2) Constriction of a blood vessel may produce increased amplitude of pulsation of the vessel, and dilatation of the vessel may actually diminish the size of the pulse wave of the vessel.

METHODOLOGY

Displacement transducers were connected in series to a calibrator and a multichannel recorder. The pick-ups were placed over the artery and held in position with a rubber band. Skull measurements were taken to identify the

point of contact between the transducer and skin so that the transducers could be reapplied to the same area in repeat tests on the same individual. The pressure used in holding the transducers in place was calibrated and, if this was a repeat test, the previous pressure was duplicated.

The drugs used in the study included sedatives, analgesics, vasodilators, vasoconstrictors, antihistamines, and hormones.

A patient being studied received a medication, either sublingually, orally, rectally, or hypodermically. A continual tracing was taken bilaterally of either the supraorbital, temporal, or the occipital arteries. The patient was asked to indicate the moment when he began to feel the effect of the medication (increase in pain, decrease in pain, head flush, and so on), and the tracings were observed for any change which occurred in the pulse wave contour.

RESULTS

Sedatives and analgesics. Various analgesics (aspirin, salicylamide) and various sedatives (sodium phenobarbital and sodium Amytal) were used separately and in combination (Fiorinal), but none had any noticeable effect on the pulse wave, even in cases where the headache was altered by these medications.

Antihistamines. These do not produce any effect on the pulse wave amplitude or on the pulse rate of headache or headache-free individuals.

Nicotinic acid. This has a marked effect on the pulse wave pattern. This effect can be either increased amplitude of pulsation or decreased amplitude of pulsation.

Histamine diphosphate. This produces a vascular reaction and a pulse volume tracing similar to that of nicotinic acid.

Ergot. Ergot, in the main, diminishes the amplitude of pulsation, but it is quite capable of increasing the amplitude of pulsation in patients. As indicated above, this increase in amplitude does not necessarily imply vasodilatation, nor does diminution in amplitude necessarily imply vasoconstriction.

Epinephrine. Epinephrine has a

TABLE 1
MIGRAINE

Medication	*Dose and Route of Administration*	*No. of Patients Tested*	*Time Variation for Subjective Response (Min.)*	*Time Variation for Objective Response (Min.)*	*Pulse Wave Response*
D.H.E.-45	1 cc. intraven.	34	3.15–8.12	4.22–10.00	25 dimin. amplit. 9 inc. amplit.
Gynergen	1 cc. intramusc.	17	5.49–12.56	6.55–15.40	15 dimin. amplit. 2 inc. amplit.
Epinephrine	0.5 cc. subcut.	4	0.50–2.10	1.46–3.35	Inc. ampl.
Caffeine suppositories	100 mg. caffeine rectal	6	——	——	——
Ergotamine tartrate suppositories	2 mg. ergot. tartr. rectal	8	18.50–27.20	21.30–29.50	5 dimin. amplit. 3 inc. amplit.
Ergotamine tartrate suppositories	4 mg. ergot. tartr. rectal	3	17.05–22.20	19.45–28.15	Dimin. amplit.
Cafergot suppository	2 mg. ergot. tartr. 100 mg. caffeine rectal	9	16.40–23.20	18.25–25.10	8 dimin. amplit. 1 inc. ampl.
Cafergot tablets	2 mg. ergot. tartr. 200 mg. caffeine P.O.	15	19.00–42.00	21.30–48.40	11 dimin. amplit. 4 inc. ampl.

TABLE 2
TENSION

Medication	*Dose and Route of Administration*	*No. of Patients Tested*	*Time Variation for Subjective Response (Min.)*	*Time Variation for Objective Response (Min.)*	*Pulse Wave Response*
Sodium luminal	2 gr. intramusc.	5	4–12.20	——	——
Sodium Amytal	3¾ gr. intraven.	3	0.45–1.12	——	——
Nicotinic acid	100 mg. intramusc.	42	3.30–5.15	4.10–8.42	31 incr. amplit. 11 dim. ampl.
Fiorinal	2 tablets P.O. Sandoptal 50 mg. caffeine 40 mg., A.S.A. 200 mg. Acetophenetidin 130 mg.	4	22–35	——	——
Aspirin	10 gr. P.O.	5	18–42	——	——

marked effect on the pulse wave contour, increasing the pulse rate and also the amplitude of pulsation.

Caffeine. Caffeine by itself has no effect on the pulse wave amplitude or rate. However, when combined with ergot, it acts as a potentiator and increases the speed with which ergot produces a vascular effect.

CONCLUSIONS

With the use of displacement transducers, the pulsations of the major extracranial arteries can be readily observed and recorded. This produces a relatively simple means of objective testing of the effect of various drugs on the extracranial arterial system, and in headache mechanisms, it is possible to correlate objective vascular changes with subjective manifestations.

According to our pulse volume readings, analgesics and sedatives have little or no effect on the extracranial arterial system. Nicotinic acid has a marked reaction on the extracranial arteries and may increase the amplitude of pulsation of a vessel or may diminish the amplitude. With vasoconstrictor drugs the intravenous route of administration produces a vascular response very rapidly. Rectal administration of ergotamine tartrate produces a vascular response in a shorter space of time than the same medication given orally. Finally, the effective use of caffeine with ergotamine tartrate is not based on the ability of caffeine to produce any vasoconstriction by itself, but apparently only on its ability to potentiate the action of ergotamine tartrate. It is also noteworthy that the subjective response to a medication is elicited before the associated vascular response can be recorded objectively.

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Cingulate Gyrus Seizures

Correlation of Electroencephalographic and Behavioral Activity in the Cat

Orlando J. Andy, M.D. and Ralph McC. Chinn, M.S.

EXTENSIVE ANATOMIC STUDIES have revealed a large portion of the cingulum to originate in the limbic cortex and terminate in the subiculum, presubiculum, and possibly hippocampus proper.[1–4] This anatomic connection has been proposed to serve as one of the main links, anatomically and possibly physiologically, in a circuit consisting of hippocampus, fornix, mammillary body, anterior nucleus of the thalamus, limbic cortex, cingulum, and back to the hippocampus.[5,6]

It is postulated that this circuit or system "elaborates the function of central emotion as well as participates in emotional expression."[5] With reference to this hypothesis, one may further entertain the possibility of a cingulate gyrus seizure contributing to some of the psychic changes manifest in psychomotor seizures. It has been proposed previously that an epileptogenic focus originating in the cingulate gyrus cortex may induce a convulsive diathesis, by way of the cingulum, throughout the hippocampal system.[7]

In this study, therefore, an attempt was made to correlate the electroencephalographic and behavioral changes occurring during a seizure induced by stimulating electrically the anterior and posterior limbic cortex (primarily in its supercallosal portion). In order to appreciate the significance and possible role played by the supercallosal portion of the limbic cortex in this circuit, the results have been compared with those obtained from comparable stimulations of the hippocampus and other structures within the limbic system.

In this paper, the terms anterior and posterior cingulate gyrus refer to the areas designated as anterior and posterior limbic cortex in the cat by Rose and Woolsey.[8] Hippocampal formation refers to the hippocampus (cornu Ammon's), facia dentata, subiculum, taenia tecta, and regio retrobulbaris, as stated by Rose for the Ammon's formation.[9]

METHOD

Nineteen freely moving unanesthetized cats were employed for this study. Sub-

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FIG. 1. Medial view of cat's brain. Composite representation of points through which both electrical stimulation and recording were made in the cingulate gyrus. In 63 of the illustrated 99 points, comparable areas were simultaneously investigated in the opposite hemisphere. Large bars represent triple needle electrodes oriented transversely and the smaller bars, triple needle electrodes oriented longitudinally. Most of the points are located within 1 to 2 mm. depth from the cingulate cortical surface. Numbers identify the experiments.

cortical triple stainless steel Hess electrodes were inserted to various subcortical structures and screw electrodes to the dura overlying specific cortical areas. The scalp was injected with 0.5 per cent Novocain to facilitate the insertion of electrodes. Electrodes have a 1 mm. bare tip, a diameter of 0.5 mm. which tapers to a point at the end, and an interelectrode distance of 1½ mm. Bipolar stimulation and recordings were made with a Grass square-wave stimulator and electroencephalograph, respectively. In four experiments a condensor type discharge with a 3.0 millisecond time constant was employed. Over 95 per cent of the stimulations were carried out with a square-wave pulse of 1.0 millisecond, frequency of 30 per second, and duration of five seconds. Voltage ranged from one to six volts, with the majority of the stimulations being carried out within a range of one and a half to three volts. Electrical and behavioral changes of the freely moving animal were recorded simultaneously with a Grass electroencephalograph and movie camera, respectively. Following the completion of each experiment, the brain was perfused with normal saline, followed by a mixture of 40 per cent formalin solution containing potassium ferrocyanide. Serial histologic sections were then made of all brains and stained with thionine. All points of stimulation and recording were then localized histologically. A total of 162 different points in the cingulate gyrus were investigated, from which 498 after-discharges were elicited. Figure 1 shows a composite orientation of 99 of these points. The remaining 63 points are in a more or

less comparable location in the opposite hemisphere which is not illustrated. Most of these points were oriented in the supercallosal portion of the cingulate gyrus and primarily concentrated in the more anterior and posterior regions. Thirty-six of the 162 points were studied with the triple needle electrodes oriented transversely. Their results were no different from the longitudinally oriented electrodes.

RESULTS

Spontaneous cingulate gyrus electrical activity does not have any characteristic appearance such as is found in the hippocampal resting activity. In contrast, it is made up of a low voltage, 25 to 50 microvolt beta frequency activity, with a mixture of occasional intermediate fast and five or six per second waves. The fast activity is often seen riding the slower waves. Spindles associated with sleep appear very readily in the cingulate gyrus, in addition to other cortical areas. The spindles have a frequency of 12 to 13 per second, occur in bursts, and have a maximum amplitude of 50 to 400 microvolts. These rhythms appear simultaneously in other cortical and subcortical structures, but there is no apparent definite synchrony. Activation of the animal with auditory or other forms of sensory stimulation causes a disappearance of the "sleep" spindles, with an appearance of a low voltage fast activity just as is frequently observed in other cortical areas.[10] In contrast, the hippocampal spontaneous activity and activation pattern are remarkably different from that of the cingulate gyrus (figure 2). Note the well organized high voltage 15 per second activity appearing in the hippocampus, in contrast to the

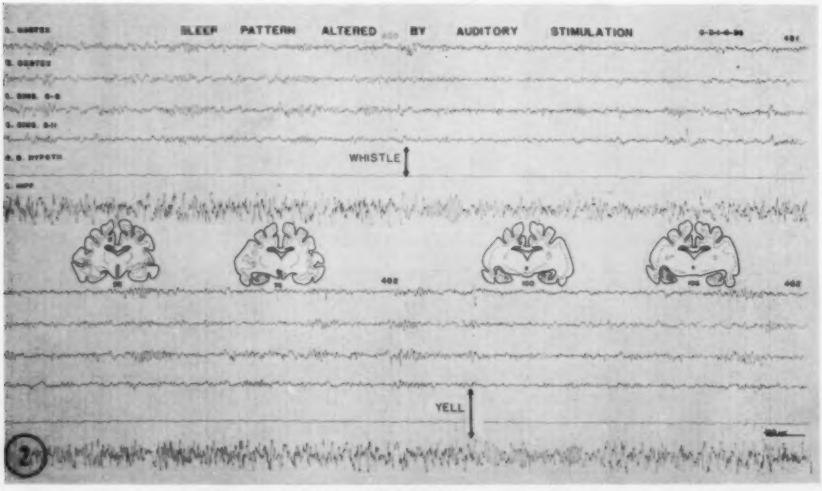

FIG. 2. Forty seconds of continuous electroencephalographic recording while the cat is sleeping. No gross alteration of the cat's attitude was observed during the auditory stimulation. In spite of this, there is a definite disappearance of the 12½ to 13 per second spindles in the cortex and cingulate gyrus. Note the appearance of 15 per second sharp waves in the hippocampus, which is in marked contrast to the activation pattern in the cingulate gyrus. (Sections 38 and 100 are left cingulate gyrus. Section 74 is right dorsal hypothalamic nucleus. Section 106 is left hippocampus.)

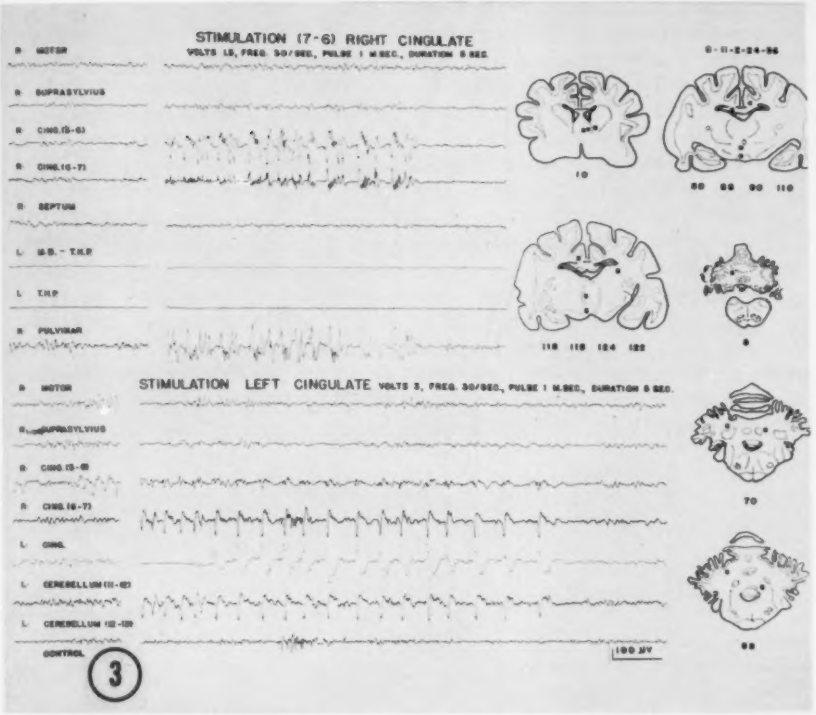

FIG. 3. Upper half: After-discharge induced in the right cingulate gyrus (section 110, insert) spreads to the right pulvinar, but not to the septum or mammillary body. Lower half: Note presence of after-discharge in cingulate gyrus bilaterally with spread to the left cerebellum (section 88, insert) and simultaneous lack of discharges in cortex. (L.M.B.—left mammillary body; T.H.P.—habenulopeduncular tract.)

very low voltage fast activity in the cingulate gyrus.

Electrical after-discharge induced in the cingulate gyrus. With threshold stimulation, the duration of the cingulate gyrus after-discharge is invariably short in comparison to hippocampal-induced seizures. The majority of the discharges will not last more than 10 seconds. However, with suprathreshold stimulations, much longer seizures may be obtained, lasting as long as 60 seconds. This is in marked contrast to hippocampal-induced after-discharges which may last longer than 60 seconds following a threshold stimulation. The amplitude of the discharge varies from approximately 150 to 700 microvolts. The pattern of the electrical discharge is characterized by a fundamentally slow and rhythmical one and a half to four or five per second mixture of slow waves, with occasional superimposed fast spiking activity. The spiking activity which appears may vary from 20 to 30 per second. They occasionally appear in groups of two to five once or twice per second. Intermediate frequencies occur sporadically. The cin-

gulate gyrus discharge usually ends very abruptly and, in most instances, is not accompanied by a post-discharge electrical depression. The previously occurring spontaneous electrical activity immediately reappears at the end of the end of the discharge just as if no alteration of the spontaneously existing electrical phenomena had taken place in the structure.

Propagation of cingulate after-discharges to other structures. A cingulate gyrus after-discharge induced in one hemisphere invariably is propagated to the homologous areas in the cingulate gyrus of the opposite side. Transcallosal propagation to the opposite cingulate gyrus appears to be more readily attained than propagation between the anterior and posterior cingulate gyrus areas of the ipsilateral hemisphere. Motor cortex is very infrequently implicated, whereas sensory areas are often involved. The various subcortical structures studied for propagated activity from cingulate gyrus after-discharge are the basal ganglia, the thalamus, the hypothalamus, mesencephalic structures, and the cerebellum. In general, following threshold stimulation, propagated activity to these various distant structures was very meager and occasionally revealed a predilection for unsuspected distant sites (figure 3). An after-discharge elicited in the cingulate gyrus

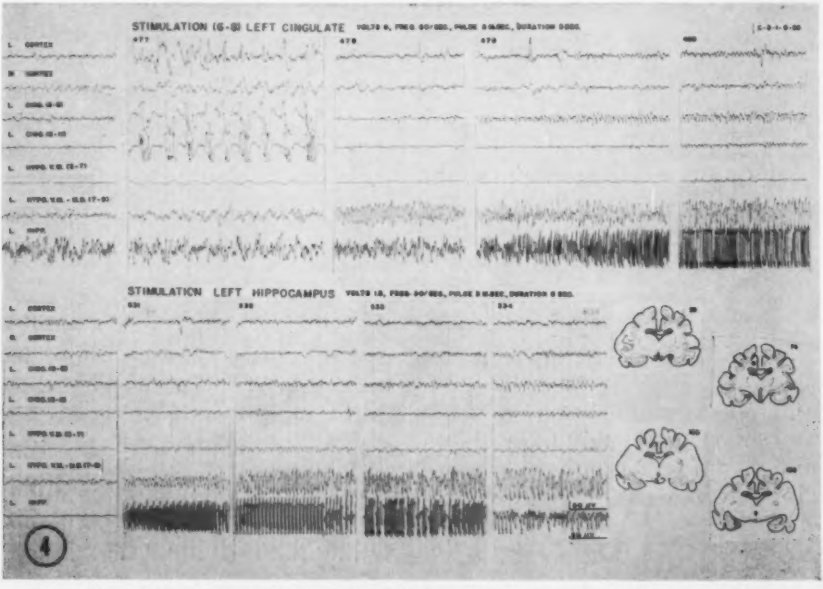

FIG. 4. Upper half: Suprathreshold stimulation of the left cingulate gyrus (section 74, insert) induces an after-discharge with eventual activation or spread to the hippocampal system, which in turn induces a propagated or spreading discharge in the cingulate gyrus as shown in the later half of the illustration. The lower half of the figure shows striking similarity between a hippocampal discharge locally induced (section 106, insert) to that occurring in the later half of the after-discharge induced by stimulating the left cingulate gyrus (Section 58 is L. Hypo. V.M.—left ventromedial nucleus of hypothalamus; section 100 is L.M.B.—left mammillary body).

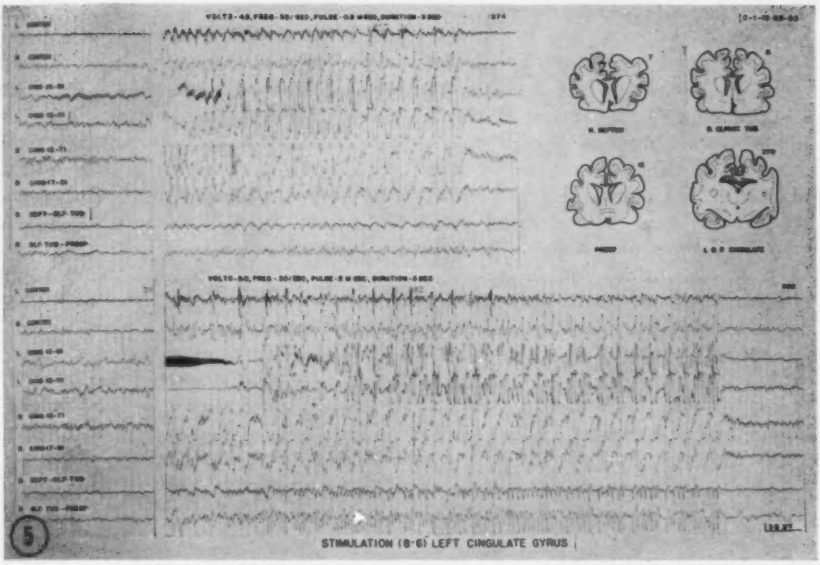

FIG. 5. Suprathreshold stimulation is necessary to produce a spread of the cingulate gyrus discharge to distant structures (left and right cingulate gyrus, section 270). Note the change in pulse duration which was necessary (0.5 to 2 mm. per second) in order to produce a spread of the cingulate discharge to the right septum (Sept.), olfactory tubercle (olf. tub.), and preoptic area (preop.) (sections 7, 6, and 12, inserts). No changes in behavior were noted.

will, in many instances, propagate to the cortex or other subcortical structures before being propagated to the hippocampus. In most instances it requires a stimulus strength much above threshold in order to have a discharge propagate to the hippocampal system (figure 4). Following a threshold stimulation of the cingulate gyrus, the electrical after-discharge definitely implicates the pulvinar nucleus, whereas the hippocampal system, as represented by the septum and mammillary body, is not involved (figure 3). It is of added interest to note that the cortex is involved (gyrus supersylvius medius) before the region of the septum, olfactory tubercle, and the preoptic area (figure 5). The latter structures are involved only after suprathreshold stimulation. The pattern of the propagated activity varied according to the structure under investigation In most instances the propagated activity to the cortical surface more or less mimicked that found in the cingulate gyrus. However, propagated activity to the various subcortical structures showed more variation in that it occasionally mimicked the cingulate (figure 3) gyrus activity, and in other instances more or less presented a discharge pattern which had no resemblance (figure 5) to that simultaneously existing in the cingulate gyrus. In several experiments, consequent to inducing cingulate gyrus afterdischarge with suprathreshold stimulation, it was possible to activate or induce a self-sustained hippocampal dis-

charge (figure 4). The reverse was never observed.

Threshold stimulation of the cingulate gyrus, in contrast to stimulation of the hippocampus, in most instances will not produce an activation pattern in the overlying cortical activity. In fact, stimulation of the cingulate gyrus with a resulting bilateral after-discharge will often be accompanied simultaneously by sleep spindles in other brain structures. Sleep spindles will rarely disappear during a bilateral cingulate gyrus seizure. In contrast, such spindles will invariably disappear in the presence of a hippocampal-induced after-discharge. A rare example of preserved spindling in the presence of a hippocampal system after-discharge is seen in figure 6. It is interesting to note that, in a series of alternate stimulations between the cingulate gyrus and hippocampus, these spindles occur more frequently during the cingulate gyrus after-discharge than during the hippocampal discharge. Propagated discharges from an induced hippocampal seizure or septal seizure appear to involve the cingulate gyrus more readily than vice versa. It has been of further interest to find that, in a few experiments in which simultaneous recordings were made between the anterior and posterior cingulate gyrus during a hippocampal-induced seizure, the slow seven to eight

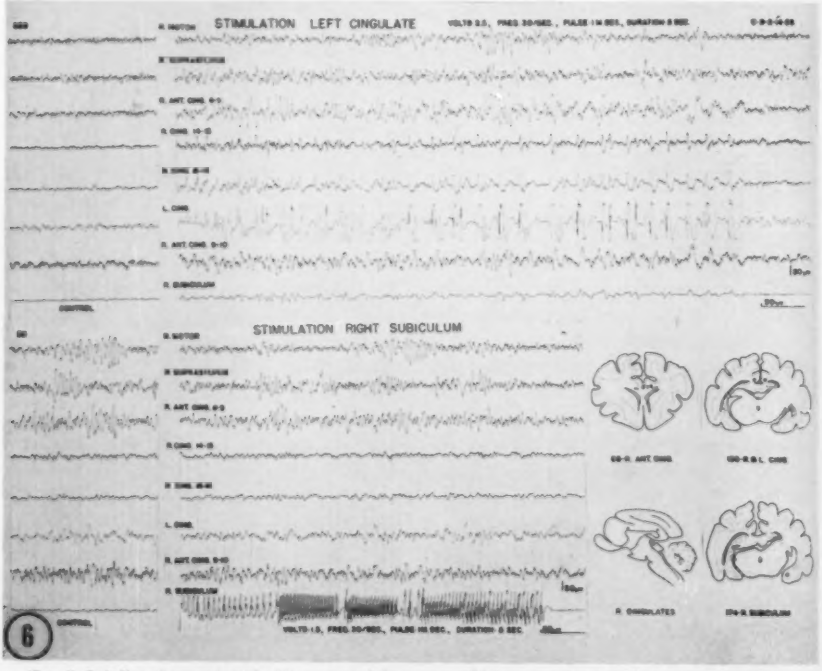

FIG. 6. Spindles of approximately 12 per second frequency, which are associated with sleep, appear during an after-discharge in the cingulate gyrus bilaterally. Note the control record representing "activation pattern" existing just before the stimulation of the left cingulate gyrus (section 150, insert).

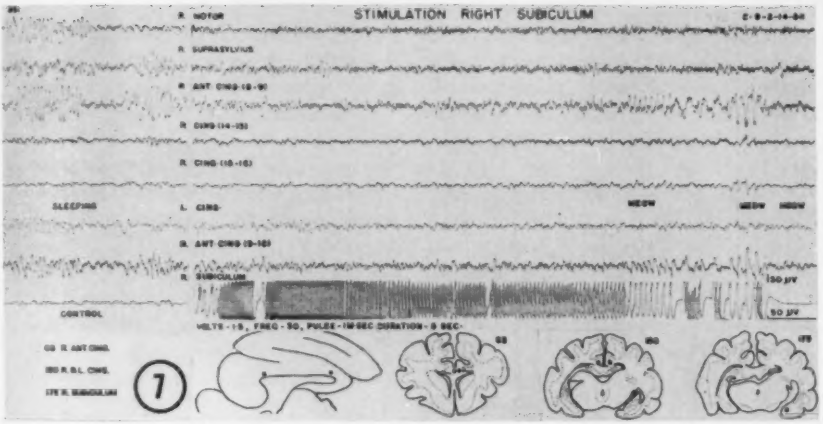

FIG. 7. Stimulation of right subiculum (section 175, insert) induces a discharge with an associated loss of sleep spindles and awakening of the animal in the later phase of the after-discharge. Note the transmission of 7 to 8 per second activity to the right anterior cingulate (section 68, insert) and complete lack of the similar activity being propagated to the right and left posterior portions of the cingulate gyrus (section 150, insert).

per second activity implicated the anterior cingulate region more readily than the posterior (figure 7). Figure 8 demonstrates activity being much more readily propagated between the hippocampal system and the anterior cingulate gyrus in contrast to the hippocampal system and the posterior region of the cingulate gyrus.

The influence of a cingulate gyrus after-discharge upon a hippocampal discharge and vice versa was studied relative to their seizure duration (figures 9, 10, and 11). As one can readily observe from the figures, a discharge in either one of the structures has no apparent effect upon the duration of the discharge. The apparent increase in duration of the hippocampal discharge, in contrast to a decrease in the cingulate discharge found in figure 10, is not considered to be due to a reciprocal influence, since repeated stimulation of the hippocampus has been found to occasionally induce progressive facilitation or prolongation of its discharge. In experiment 13 (figure 12) an attempt was made to facilitate or prolong the duration of a cingulate gyrus seizure by preceding the cingulate gyrus stimulation with stimulation of the mesencephalic reticular system. Although stimulation of this system appeared to produce cortical activation, as described previously,[11] it did not appear to facilitate the seizure. In fact, following the course of stimulations in the mesencephalic reticular system, a few of the cingulate gyrus stimulations became ineffective.

Threshold studies. In general, the threshold stimulus necessary for inducing a cingulate gyrus discharge was a little higher than that necessary to induce a hippocampal discharge. The difference ranged between one and two volts, with other parameters remaining constant. However, in a few experiments a low voltage threshold approximating that in the hippocampus was found. In general, it has been noted that, with re-

peated stimulation, the threshold for an after-discharge in the cingulate gyrus rises more quickly and is not as stable as that found in the hippocampus. It is this factor which makes one question seriously the results demonstrated in figures 10, 11, and 12.

A relatively low voltage fast frequency injury potential occasionally develops following the insertion of needles into the limbic cortex. However, it does not progress into a well organized after-discharge pattern simulating those induced by electrical stimulation. It is of interest to note that an after-discharge, described previously as resulting from mechanical stimulation of the hippocampal system and presumably indicative of an existing low threshold, was not found in the cingulate gyrus structure.

Behavioral changes during cingulate gyrus after-discharges are surprisingly minimal. The sensory system appears to remain intact for various modalities such as visual, auditory, tactile, and painful stimulations. The occasional exaggerated response to a sensory stimulus during a hippocampal after-discharge was never seen during the cingulate after-discharge. If a cingulate discharge is induced while a cat is sleeping, in most instances there is no interruption of the behavioral or

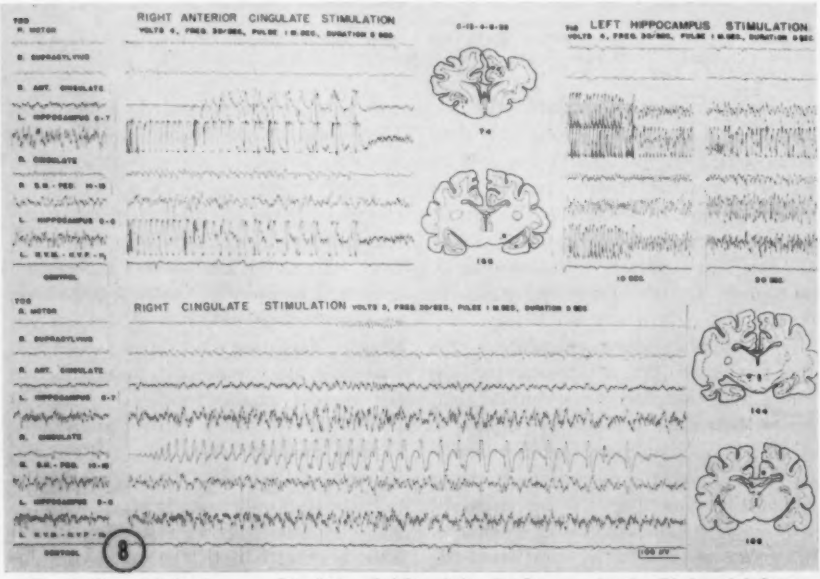

FIG. 8. Upper left hand corner: Stimulation of right anterior cingulate area (section 74, insert) induces an after-discharge with spread to the left hippocampus and relatively very minimal spread to the posterior right cingulate gyrus (section 186, insert). Lower half of figure: An after-discharge induced in the right posterior cingulate gyrus (section 186, insert) of the same animal does not implicate the hippocampus or anterior cingulate region. Upper right corner of figure: Stimulation of left hippocampus induces a marked after-discharge in the right anterior cingulate gyrus, especially during the first ten seconds of the discharge, in contrast to very little involvement of the right posterior cingulate in the same animal (L. N.V.M.—left ventromedial nucleus of thalamus; L. N.V.P.—left nucleus ventralis posterior medialis; H_1—anterior field H_1 of Forel, section 144, insert; R. S.N.—right substantia nigra; R. Ped.—right peduncle, section 166, insert).

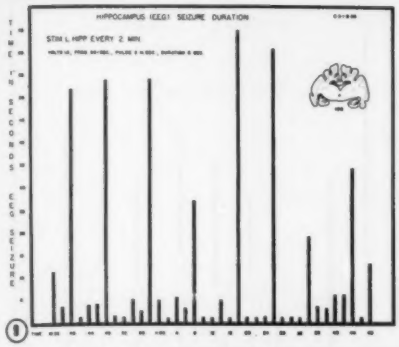

FIG. 9. Stimulation of the left hippocampus (section 106, insert) every two minutes responds with a prolonged after-discharge every eight to ten minutes.

electrical activity. However, during a hippocampal discharge the animal will invariably become aroused from sleep and look around. Meowing, which is often present at the termination of a hippocampal discharge, is not associated with a cingulate gyrus after-discharge. If the cat's tail is pinched during a cingulate after-discharge or a mouse is presented, the cat will attack very readily. However, if the discharge appears to spread to the hippocampal, amygdaloid, or other subcortical brain structures, there may be an alteration of the expected response. There is no alteration of motor activity during a cingulate gyrus after-discharge. The animal is capable of lapping milk and carrying out other motor functions with no incoordination. There are no tonic or clonic movements. Motor movements were only seen in a few instances during a discharge which was induced with suprathreshold stimulation. The associated discharge had invariably propagated to distant subcortical and cortical structures. Contralateral or ipsilateral turning of the head was not evident with stimulation of the cingulate gyrus unless a suprathreshold stimulus was employed. Urination, defecation, pupillary dilatation, and altered respiration and pulse rates were rarely observed.

DISCUSSION

Seizures induced in the cingulate gyrus apparently contribute very little to the behavioral and psychic changes associated with hippocampal and amygdaloid after-discharges, as observed by numerous investigators.[7,12–17] Emotional changes, such as rage or alteration of a physiologically existing aggressive behavior, were not observed with cingulate gyrus stimulation or after-discharges as was found in stimulation of other regions in the limbic system. In one animal extremely aggressive behavior was expressed toward the experimenter in the form of hissing, biting, and scratching. It is pertinent to note that, during a hippocampal-induced after-discharge, this animal became quite docile and was easily handled. However, during a cingulate gyrus-induced after-discharge, the animal's aggressive behavior was not altered.

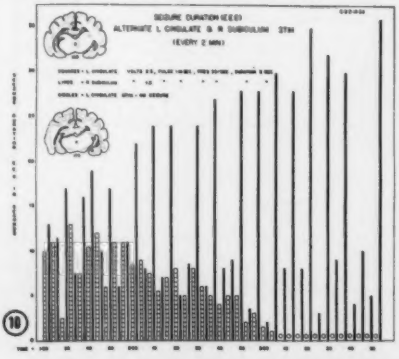

FIG. 10. Alternate stimulation of left cingulate gyrus (section 150, insert) and right subiculum (section 174, insert).

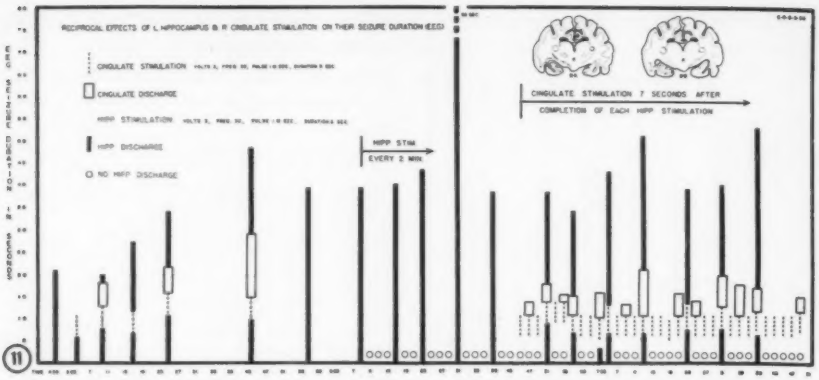
RECIPROCAL EFFECTS OF L HIPPOCAMPUS & R CINGULATE STIMULATION ON THEIR SEIZURE DURATION (EEG)
CINGULATE STIMULATION
CINGULATE DISCHARGE
HIPP STIMULATION
HIPP DISCHARGE
NO HIPP DISCHARGE
HIPP STIM EVERY 2 MIN
CINGULATE STIMULATION 7 SECONDS AFTER COMPLETION OF EACH HIPP STIMULATION

FIG. 11. Right cingulate gyrus stimulation (section 94, insert) with subsequent after-discharge does not appear to alter the duration of a hippocampal-induced after-discharge (section 99, insert).

Somatic changes, such as head movement, facilitation or inhibition of motor-induced activity, and apparent arrest of the walking[18] animal, were not observed in this series of experiments. Controversive head movements and arrests of spontaneous activity were only observed during an excessively strong stimulus or an after-discharge produced by suprathreshold stimulations.

No alteration of respiration, pupillary dilatation, vocalization, pilo-erection, or urinary bladder or bowel activation was observed in these animals, as was commonly observed in the anesthetized or partially anesthetized preparation.[19–24]

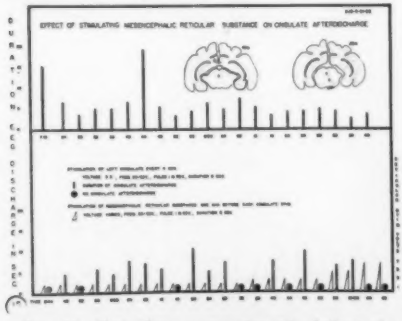

FIG. 12. Forty-three consecutive stimulations of the left cingulate gyrus (section 196, insert) every four minutes. In the last half of the series the mesencephalic reticular substance (section 204, insert) was stimulated one minute before each of the cingulate stimulations.

After-discharges from the cingulate gyrus are not associated with suppression or inhibition of behavioral or electrical activity; neither was there evidence to substantiate a possible role of activation. Recently various investigators have tried to include both the cingulate gyrus and the hippocampus in the activating system of the brain. There is a marked contrast between an after-discharge in the cingulate gyrus, which is accompanied by continuous sleep spindles in the electroencephalogram and an undisturbed attitude, and a hippocampal-induced discharge, which is accompanied by electrical activation in the encephalogram and awakening of the animal with meowing at the termination of the seizure. Electrically, the limbic cortex appears to behave very similarly to the cortex over the convexity of the brain, which is markedly different from that found in the hippocampus.

The role that the cingulate gyrus plays

in the circuit previously proposed[5,6] appears to be very subtle and should certainly undergo much more extensive investigation before any definite conclusions can be drawn. In accordance with these studies, one is inclined to believe that the link from the cingulate gyrus to the hippocampus is a weak one functionally.

SUMMARY

1. In 19 adult cats prepared under local anesthesia, the supercallosal portion of the cingulate gyrus was stimulated electrically, and the electroencephalographic and behavioral changes were recorded simultaneously on a Grass electroencephalograph and movie camera, respectively. Results are contrasted with those obtained from hippocampal stimulation and after-discharges.

2. Spontaneous electrical activity in the limbic cortex appears similar to that found in other cortical areas and does not simulate that found in the hippocampus.

3. Twelve to 13 per second spindles associated with sleep appear very readily in the cingulate gyrus and may or may not be synchronous with those appearing in other cortical areas such as the motor and sensory regions.

4. Sleep spindles appearing in the cortex during a cingulate gyrus after-discharge remain unaltered, whereas they invariably disappear during a hippocampal discharge.

5. The electroencephalographic pattern of a cingulate gyrus after-discharge is characterized by waves of relatively slow frequencies with occasional superimposed bursts of 20 to 30 per second activity. Its pattern tends to simulate discharges obtained from other cortical areas.

6. The duration of a cingulate after-discharge is much shorter than that of a hippocampal discharge.

7. Stimulation of the limbic cortex produces no apparent activation of the cortical electrical activity as is found with hippocampal stimulation.

8. A cingulate gyrus seizure of one hemisphere invariably propagates to the opposite side. Motor cortex in contrast to sensory cortex is seldom involved. Threshold-induced seizures in the hippocampus propagate to the cingulate gyrus much more readily than vice versa.

9. The threshold for inducing a cingulate gyrus discharge is greater than that necessary for a hippocampal-induced discharge.

10. Hippocampal after-discharges have no apparent influence upon the duration of cingulate gyrus discharges or vice versa.

11. Behavioral changes during a cingulate gyrus seizure are very minimal, unless suprathreshold stimuli are used.

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■ When a tumor exists, pressing upon the anterior surface of one of the crura, cerebelli and upon the insertion of the trigeminal nerve (see Fig. 25) if it causes paralysis, it is in the same side of the body. I have collected fourteen cases of this kind, all having the same features, which are incomplete paralysis in the side of the lesion, no anaesthesia (except in one case) and frequent fits of vertigo. Now, as to the explanation of this kind of paralysis, we will say, that it is either the result of the destruction of some conductors employed in voluntary movements (to regulate them or to act otherwise) or of the irritation of certain nervous fibres in the peduncle itself, or near it. Were the first hypothesis the true one, we should find that a destruction of the whole peduncle causes paralysis in the corresponding side only, or in it and in the other one, and not in this other alone; but there are several cases in which there has been, with such an alteration, a paralysis in the opposite side only. We should find, also, that alterations of the parts by which the crus cerebelli communicates with the muscles reproduce a paralysis in the same side of the body, together with a paralysis in the opposite side. But this is not what is observed. I have collected more than thirty cases of alteration in a lateral half of the pons Varolii and medulla oblongata, in many of which the lesion extended to the crus cerebelli, and in all the paralysis was in the opposite side only. For instance, in a case of Dr. Anna, which I have related (see Case 38, Lecture VII), the whole connection of the right crus cerebelli with the right half of the medulla and of the pons was destroyed, and the paralysis existed only in the left limbs.

C. E. Brown-Séquard in *Lectures on the Physiology and Pathology of the Central Nervous System,* published in 1860.

CASE REPORT

Homonymous Hemianopsia Following Interruption of the Parieto-occipital Vein

Keasley Welch, M.D.

THE PARIETO-OCCIPITAL VEIN is a large vessel which leaves the surface of the cortex and enters the superior sagittal sinus at about the level of the parieto-occipital fissure. Its components are from both the medial and convex surfaces of the cerebral hemisphere. Although not singled out for description in most anatomic texts, it is well described by O'Connell in his study of the superior cortical veins.[5]

Occlusion of this vein in human subjects results in homonymous hemianopsia which is likely to recover. The evidence for this is that contralateral hemianopsia has followed on each of three occasions when surgical situations have required that the vein be divided. During one of these operations, the patient was awake and able to cooperate in visual field examination; he was found to have developed an homonymous hemianopsia 35 minutes following occlusion of the vessel.

CASE REPORTS

Case 1. A nine year old girl had previously had a ventriculogram and unfruitful suboccipital exploration elsewhere. Studies revealed a tumor in the posterior part of the third ventricle.

Operation. On June 25, 1952 the tumor was approached by Dandy's method through a left parietal bone flap. A single large vein, passing from the parieto-occipital region to the superior sagittal sinus, required division. The tumor, an astrocytoma, was largely cystic. After removal of cyst wall and biopsy of the solid part, the wound was closed. No vessels were divided in the depth of the wound.

Course. The child remained unconscious for three days, the unconscious state resembling normal sleep. When she awoke there was some incoordination and weakness of the right arm and there was right homonymous hemianopsia. By July 31, the hemianopsia had cleared to gross confrontation.

The child has continued well, and on May 18, 1954 a visual field examination showed normal findings both on the perimeter and tangent screen.

Case 2. A 23 year old woman was studied after a second subarachnoid hemorrhage. Physical examination, including visual field study, was normal. Arteriography revealed an arteriovenous aneurysm in the medial part of the right parietal lobe.

Operation. On November 27, 1953 the lesion was excised. The approach was made through the sagittal fissure, with the right side of the head in the undermost position. In order to gain access within the fissure, a large parieto-occipital vein was ligated. The lesion was subcortical just above and to the right of the splenium of the corpus callosum.

Course. On the day of operation left homonymous hemianopsia was discovered. There were no other neurologic deficits. Recovery was uneventful. By November 29 she could distinguish light and dark in the left field. The hemianopsia cleared completely as demonstrated by an examination April 15, 1954.

Case 3. A 32 year old man was studied because of intense headache of five days' duration. He had experienced similar episodes 12 years and again one month before. There was papilledema and inability to look upward. On

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ventriculography there was hydrocephalus and a filling defect in the posterior part of the third ventricle.

Operation. On September 16, 1954 the lesion, an ependymoma, was approached and biopsied from the right side through the sagittal fissure, utilizing local analgesia. There was a large parieto-occipital vein passing from the surface of the brain to the superior sagittal sinus, and this was divided at 11:00 p.m. Gross visual field examinations were made at five-minute intervals after this, and at 11:35 p.m. the patient exhibited a left homonymous hemianopsia. Meanwhile no other vessels had been interrupted, the medial parietal lobe had been retracted gently, and the posterior part of the corpus callosum had been incised. After the biopsy was made, an external ventriculostomy tube was placed. At the conclusion of the procedure the patient was as well as before, with the exception of the hemianopsia which was again observed.

Course. The following morning he was drowsy; the ventriculostomy was obstructed by blood. A ventriculocisternostomy was made. He died September 18 of ventricular hemorrhage from the tumor.

DISCUSSION

The hemianopsia which frequently occurs as a result of operation within the posterior part of the sagittal fissure is well known, and some years ago it gave rise to considerable discussion. Dandy[1,2] observed it three times after the posterior sagittal approach to third ventricle and pineal tumors, and considered that retraction of the visual cortex was responsible. Fortunately Dandy's case reports include many details about the surgical operations. His published experience includes 14 cases in which the midline posterior operation was done. Of these, six cases are not suitable for consideration for one reason or another: three died, one was blind before operation, another was blind afterward, and in still another occipital lobectomy was carried out. Of the remaining eight cases, two are specifically mentioned as having been spared postoperative hemianopsia, and these two were operated upon without sacrifice of cortical veins. The three instances of hemianopsia were encountered in the remaining six cases. Two are identified in the individual case reports in the two publications.

Harris and Cairns also observed this deficit after an operation for pineal tumor and had some interesting comments about its genesis.[3] They ascribed the visual field defect which occurred to interruption of the internal occipital vein. Cairns approached the lesion through a large parieto-occipital bone flap, and divided all the superior cortical veins behind the postcentral vein on one side. The internal occipital vein drains the medial surface of the occipital lobe and empties into the deep venous system. It is difficult to understand why, in this case, the deficit should be ascribed specifically to occlusion of the internal occipital vein when so many veins were interrupted.

In reference to this hypothesis, it is important to note that, in case 1 and case 3 reported here, the internal occipital vein was not molested. It should not be thought, however, that the internal occipital vein is of no importance in this connection. No doubt its role as an alternative route for venous drainage is of great importance.

This interesting visual field defect was also observed and commented upon by Kahn,[4] who favored Dandy's idea that it was due to retraction rather than Harris' and Cairns' proposal, reference to which has already been made. It is doubtful, moreover, whether in any of these cases the cortex bordering the calcarine fissure has actually been subject to retraction. Dandy's pineal approach is rather higher and forward of the cuneus and, if bruising is to occur, it is likely not to involve the visual cortex.

O'Connell's study of the superior cortical veins is unique in precision and detail.[5] He states, "Three or more large veins passed from the surface of the parietal lobe to the sinus. In the majority of cases no vein was found passing from the occipital cortex to the sinus, although a vessel on the surface of the hemisphere in this region was always found passing forward to open into a large vein, constantly present in the lateral pa-

rieto-occipital fissure." After discussing further the relation of cortical veins to sulci, he continues, "In respect to this relationship of veins to sulci, it should finally be pointed out that in all cases a large member of the series was found leaving the surface of the hemisphere in the region of the lateral parieto-occipital fissure en route for the superior sagittal sinus." And he concludes his discussion as follows: "Lastly, it should be noted that each of the larger of the cortical veins, as it passes from the lateral surface of the cerebrum, receives a large tributary from the medial surface of the hemisphere. The largest of these medial tributaries passed superiorly in the medial parieto-occipital fissure to join the vessel found in the lateral portion of the fissure (which has already been mentioned)."

Although these observations were made on fetal brains, they conform with the usual adult situation as observed at surgical operations.

There has been an understandable reluctance on the part of anatomists to name individual superficial cortical veins. Although the deep venous system of the brain is relatively consistent in arrangement, the pattern of veins on the cortical surface is not. An assumption has even been made that no one of the superior cortical veins is important, because the collateral circulation is so well developed. Although this may be true for the superior cortical veins in the frontal region, one or more of which can be occluded without observable functional impairment, the present observations indicate that it does not apply at all to the parieto-occipital vein. The precise anatomic arrangement of this vessel explains why it cannot be sacrificed without penalty. There is ordinarily no alternative pathway to the superior sagittal sinus from the occipital lobe. This drainage is, on the convex surface, forward into the parietooccipital vein near the crest of the hemisphere and, on the medial surface, forward and upward to the parietooccipital vein. Thus a large component of the venous drainage of the occipital lobe is removed with occlusion of this vein. The collateral venous drainage is then through the inferior cortical veins to lateral sinus and from the medial surface into the deep venous system, a collateral circulation which is apparently not immediately suited to requirements at the moment of occlusion. That collateral venous drainage does later develop is shown by the recovery which can occur.

In this connection, the observation of Sorgo[6] should be mentioned. He used Naffziger's approach to remove a cerebellopontine angle tumor and divided the vein of Labbé and also inferior cortical veins in the temporo-occipital region. Following this operation there was a contralateral homonymous hemianopsia which recovered after some months.

Recovery is, indeed, an interesting feature of this particular sort of injury to the calcarine cortex or geniculocalcarine path. In the two present cases which could be followed, complete recovery was observed, but this has not occurred invariably in those cases reported in the literature.

SUMMARY

Three instances of contralateral homonymous hemianopsia have been observed to follow division of the parieto-occipital vein. In one case the vessel was divided in an alert and cooperative patient who was awake, and the visual field defect was seen 35 minutes later. Complete recovery occurred in the two patients who survived.

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CASE REPORT

Combined Trigeminal and Glossopharyngeal Neuralgia

John E. Adams, M.D.

THE SYNDROME of glossopharyngeal neuralgia is now a well-recognized clinical entity. Recurrent paroxysmal pain in the distribution of the ninth cranial nerve was apparently first described by Weisenburg in 1910.[1] It is of interest that his patient also had pain in the distribution of the fifth cranial nerve. In this instance, surgical exploration revealed the presence of a posterior fossa neoplasm.

In 1935, Peet, reporting in detail upon 14 patients with glossopharyngeal neuralgia, found that in five of them pain was also present in the distribution of the trigeminal nerve.[2] In describing the clinical picture of glossopharyngeal neuralgia, Peet made the following statement: "The symptomatology of glossopharyngeal neuralgia is remarkably uniform. The onset is without prodromal symptoms or other warning. Suddenly, while talking or swallowing, a sharp, knife-like pain shoots downward from the ear, the base of the tongue, or the posterior pharynx."

The majority of patients with glossopharyngeal neuralgia will give this distinctive description of the character and course of their pain. However, if both the fifth and the ninth cranial nerves are affected, the symptomatology may not present this uniform pattern, or the patient may be unable to describe the pain adequately. This is particularly true if the spasms of pain arise from both nerves concurrently. In such cases, the patient's description of the pain may give no clue to the presence of the associated neuralgia, and in fact may be completely misleading both as to the exact location and type of pain involved. Under these circumstances, definitive diagnosis may become extremely complex.

The following case is presented because of the difficulty with which correct diagnosis was finally established. As will be seen, effective treatment was achieved only after the patient had suffered for many years. During this time, numerous other forms of therapy, including psychiatric, were attempted without relief. This brief report is submitted in the hope that it may be of value in helping others avoid the same mistakes.

CASE REPORT

A 64 year old white married housewife first noted the onset of left facial pain in 1935, several months after extensive dental work had been done. The attacks remained essentially the same throughout the next 15 years.

From the department of neurological surgery, University of California School of Medicine, San Francisco.

According to the patient's description given to numerous consultants during this period, the pain was sudden in onset, sharp, stabbing, "twisting," and burning in type. It apparently originated in the left anterolateral neck at the level of the thyroid cartilage, and the patient described it as radiating from deep in the throat up to the roof of the mouth, then to the left side of the face, and up to the top of the head. The attacks usually lasted no longer than one to two minutes and disappeared as rapidly as they appeared, leaving a residual soreness and discomfort in the throat and left temple. The pain could be precipitated by swallowing, talking, and chewing, or by touching trigger zones in the lower lip and the lateral aspect of the nose.

During the period from 1935 to 1950 the patient had occasional remissions from this pain, the longest of which lasted for eight months. She visited numerous physicians, but treatment was usually limited to various types of analgesics. In addition, triethylene provided brief relief on one occasion. In 1945 injection of the left mandibular nerve with procaine alleviated the pain for two days. On another occasion the base of the tongue on the left side was injected with procaine, but again the relief was very temporary. The patient was also seen by several orthodontists, and extensive procedures were undertaken to alter her bite in an attempt to correct what was felt to be abnormal function of the temporomandibular joint. At one time degenerative arthritis of the cervical spine was suspected as the source of the pain, and cervical traction was tried for a prolonged period without relief. Other observers considered the patient's difficulty to be entirely psychogenic in origin, and psychotherapy was instituted without effect. During this 15-year period atypical facial neuralgia and temporomandibular arthritis were the diagnoses most frequently employed.

The author first saw the patient in 1950 and concurred in the diagnosis of atypical facial neuralgia, as did other members of the neurosurgical staff. No definitive treatment was recommended, and the patient did not return until five years later.

In February 1955 the patient was admitted to the University of California Hospital. During the preceding six months the pain had been particularly severe and a weight loss of 25 pounds had occurred. The patient's weight at the time of hospitalization was 88 pounds. She was having frequent spasms of pain. During each paroxysm she would thrust her tongue vigorously against the left pharynx, with her mouth partially open, and at the same time press her fingertips forcibly against her throat just below the ramus of the mandible. When questioned more closely as to the type of pain in her throat, she described it repeatedly as a "choking" sensation.

Because of the patient's insistent statement that the pain appeared to originate in the back of her throat, the left tonsillar fossa and the left side of the posterior pharynx were cocainized. This treatment completely relieved both the throat pain and the sense of suffocation. However, she continued to have occasional pain in the left lower face, radiating up to the left temple.

The patient was kept in the hospital for ten days. During this time the throat was procainized prior to each meal. This enabled her to eat without discomfort and she gained ten pounds. She was discharged with instructions to use a pontocaine spray to the back of her throat during the attacks of pain.

Second Admission. In May 1955 the patient was readmitted to the hospital. She had again lost ten pounds in weight and was having severe, constant paroxysms of pain. On the day following admission the left ninth nerve was sectioned through a linear left occipital incision. At the time of this procedure there was no evidence of neoplasm or obvious pathologic change. Transient loss of sensation occurred in the left tonsillar fossa and the posterior pharynx, postoperatively, but this sensory loss disappeared within ten days.

The operation was followed by complete relief of the throat pain or "sensation of suffocation," but paroxysmal pain involving the second and third divisions of the trigeminal nerve continued to occur. It therefore became apparent that, in addition to the throat pain, we were dealing with paroxysmal left facial pain characteristic of trigeminal neuralgia, originating in the third division, with overflow into the second and occasionally into the first division of the fifth cranial nerve. The pathway of this pain had previously been obscured by the concurrent spasms which arose from both the fifth and the ninth cranial nerves and by the patient's inability adequately to describe the type and location of her pain.

On the twelfth postoperative day, the left third division was injected with 1 cc. of absolute alcohol at the foramen ovale. However, the alcohol undoubtedly penetrated into the ganglion since immediate complete anesthesia over all three divisions of the left fifth nerve and a transient palsy of the left external rectus muscle occurred.

Since this injection, the patient has been entirely free of pain. She has gained 40 pounds, and at the present time is completely asymptomatic.

SUMMARY

A case of concurrent trigeminal and glossopharyngeal neuralgia, mistakenly diagnosed as atypical facial neuralgia over a prolonged period, is reported. The patient's inability to indicate the exact anatomic distribution of the pain and the fact that the pharyngeal and tonsillar pain was described as a "choking" sensation, together with the complexity of the clinical picture, misled many examiners including the author in the attempt to reach a definitive diagnosis.

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■ Before entering into the subject of the influence of the nervous system upon the functions of organic life, it is necessary to state what are the effects of the section and of the galvanization of the cervical sympathetic nerve. I hope I may be allowed to fix some dates of publication of the principal discoveries in this respect. Prof. Cl. Bernard published the results of his first researches on the effects of the section of the cervical sympathetic nerve in 1851, and in the bginning of 1852. The only great fact announced in these publications was, that this section was constantly followed by a considerable afflux of blood in the parts of the head to which the sympathetic goes. Led by experiments that I had made several years before, with my friend Dr. Tholozan, on the influence of nerves on blood vessels, I understood at once that the fact discovered by Prof. Bernard was due to the paralysis of the blood vessels after the section of the sympathetic; and I thought that if this view were right, I should find galvanization of this nerve producing the reverse of the effects of the section. The experiment being made, I found, as I had foreseen, that the blood vessels contracted, and that the quantity of blood and the temperature diminished. The date of my first publication is August 1, 1852. Three or four months afterwards, Prof. Bernard not knowing what I had done, announced to the Societe de Biologie (in October and November, 1852) that he had seen galvanization of the sympathetic nerve diminish the quantity of the blood and the temperature. In February 1853, a very able English physiologist, Dr. Augustus Waller, being unaware of what I had published, and of the more recent paper of Prof. Bernard, announced to the Academy of Sciences of Paris, that he had found the galvanization of the sympathetic nerve producing effects opposite to those of its section; and he gave the same theory that I had already proposed – a theory which has since been admitted by almost all the physiologists who have written on this subject.

C. E. Brown-Séquard in *Lectures on the Physiology and Pathology of the Central Nervous System*, published in 1860.

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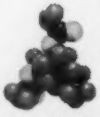

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JANUARY 1957

Two New Special Courses To Precede Academy Meeting

Two new special review courses have been programmed to precede the annual AAN meeting to be held at the Sheraton Plaza Hotel in Boston (April 25-27). One is on pain, its mechanisms and management (Charles A. Kane, director) which will be given April 22; the other is on the brain and its communication systems (G. H. Glaser, director) which will be held April 23. The course on neurochemistry (D. B. Tower, director - April 23), which was introduced and proved so popular last year, will be repeated and include recent advances. Also offered again are special courses in neuropathology (Fae Y. Tichy, director - April 22); episodic disorders of the nervous system (Francis M. Forster, director - April 23); current advances in neurology Joe R. Brown, director - April 24); and neuropharmacology (Maynard M. Cohen, director - April 24). The latter course will include consideration of tranquilizers and hallucinogens, antiparkinsonian and antispastic drugs, anticonvulsants and analgesics, general anesthetic and autonomic drugs....The fees for all courses are $10 for junior members; $15 for fellows, active, and associate members; and $20 for nonmembers, except for the course in neuropathology, which is $15 for junior members; $20 for fellows, active, and associate members; and $30 for nonmembers. All communications and inquiries should be forwarded to Dr. A. B. Baker, Chairman, Committee on Special Courses, University Hospital, University of Minnesota, Minneapolis 14....

Bennett, Denny-Brown and Szent-Gyorgyi To Spearhead AAN Muscle Symposium in Boston

The Symposium of Inquiry to be presented by the AAN Research Committee as part of the Academy's program in its forthcoming annual meeting (Boston, April 25-27) will concern muscle and its disorders. Among the participants are Washington's (Seattle) electron microscopist and professor of anatomy, G. Stanley Bennett; Harvard's professor of neurology, Derek Denny-Brown; and Nobel Laureate, Szent-Gyorgyi. The symposium will center on the anatomy, physiology, chemistry, and pathology of muscle in health and disease, and will consider new avenues for future research in this field. The presentations and discussions will be recorded, edited, and later published....Presiding at the sessions will be Augustus S. Rose, chairman of the AAN Research Committee and creator of its symposia of inquiry. When queried about the coming muscle symposium, Dr. Rose said, "It is excitingly apropos, coming at a time of mounting scientific interest in muscular dystrophy and related conditions."

AAN Colloquium On Neurochemistry The newly created section on neurochemistry in the AAN will hold its first colloquium in separate sessions at the AAN annual meeting in Boston, Friday, April 26. Among the speakers are: Donald B. Tower, on the development and future of neurochemistry; Roger J. Rossiter, lipid and nucleic acid metabolism in brain; Irwin Wilson, aspects of nerve gas poisoning; and Henry McIlwain of Maudsley Hospital, on maintenance of the composition of isolated cerebral tissues. Also, shorter scientific papers on neurochemistry will be presented concurrently at the time of the scientific sessions, April 25 and 26....For the general program of the AAN meeting, titles and abstracts (not to exceed 150 words) should be forwarded to the scientific program director, Dr. James L. O'Leary, Division of Neurology, Washington University, School of Medicine, St. Louis 10, Mo....Titles and 150-word abstracts of demonstrations or exhibits should be sent to Dr. Louis D. Boshes, Dept. of Neurology and Psychiatry, Northwestern University Medical School, 303 E. Chicago Avenue, Chicago 11, Illinois.

International Neurologist, Robert Wartenberg, Dies A symbol of world neurology, Robert Wartenberg, died from multiple coronary and cerebral vascular insults in San Francisco on November 16, two hours after he had completed his fourth book, "Neuritis, Sensory Neuritis and Neuralgia"....Born in Russia 69 years ago and educated in Germany, he came to this country in 1936 as a refugee from Naziism. He joined the University of California faculty as lecturer and research associate and nine years later became clinical professor....Robert Wartenberg possessed a unique flair for both precise observations and colorful expressions which reflect well in his teachings and writings. Rugged in appearance and action, he was a natural champion for causes, his two favorites being the AAN and the U.S.A. His compelling personality and forthright expressions served as powerful weapons in the early development of the AAN. His services as a member of the editorial board of NEUROLOGY were invaluable. While by nature a beloved colleague and indulgent friend, he could become either blunt or sharp when confronted with an appropriate challenge....Robert Wartenberg was best known as an author of four books and 158 articles, as a bedside teacher with few parallels, and later as a dominant critic of neurologic literature. His influence was world wide; his book, "Examination of Reflexes," was translated into six languages; and he was record-holder of honorary memberships in foreign neurologic societies....Dr. Wartenberg is survived by his widow, the former Baroness Isabel von Sazenhofen.

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Neuropathologists Organize Special Symposia for Brussels Under the general auspices of the First International Congress of Neurological Sciences (Brussels, July 21-28, 1957), the Third International Congress of Neuropathology will have two special symposia. The first symposium, July

25, is on the pathology of ionizing radiation, with Webb Haymaker serving as topic director; the second, July 26, under the direction of M. Gozzano (Italy), is on the cytology of the nerve cell. These two symposia are program additions to the neuropathologists' major topic: "The Pathology of Demyelinating Diseases" (Armando Ferraro, topic director)....Under the direction of A. Earl Walker, the program of the International League Against Epilepsy will highlight recent advances in the field: clinical (Macdonald Critchley); EEG (F. A. Gibbs); automatisms (Wilder Penfield); pathology (H. Gastaut); and a report on the Second International Colloquium on Temporal Lobe Epilepsy....The First International Congress of Neurological Surgery is to present three principal themes: Therapeutic Applications of Hypothermia (E. H. Botterell); Supratentorial Angiomas (H. Olivecrona); and Stereotaxic Methods (L. Christophe). The first two of these presentations will be given in conjunction with the Fifth Symposium Neuroradiologicum.

Among the officers of the Neurosurgical Congress are Sir Geoffrey Jefferson, president; A. E. Walker, vice-president; M. David, secretary-general; and W. B. Scoville, assistant secretary-general....Special features (Saturday, July 27) to be held under the auspices of the Council for International Organizations of Medical Sciences are roundtable discussions on the future of neurology, under the direction of Sir Francis Walshe....Attention is called to the fact that travel and hotel reservations may become scarce. Further information and copies of the official information brochure on the Congress may be obtained from Dr. Pearce Bailey, Secretary of the Committee for the U. S. (Sixth International Neurological Congress), National Institute of Neurological Diseases and Blindness, Bethesda 14, Maryland.

International Conference To Be Held in Caracas

About 50 of the world's leading neurologic scientists are expected to participate in a symposium on "The Submicroscopic Organization and Function of Nerve Cells." As announced by the Institute's Director, H. Fernandez-Moran, the symposium is being organized by the International Society for Cell Biology and sponsored by the Venezuelan government. Its purpose is to assemble a leading group of scientists in electron microscopy, electrophysiology, and biochemistry, "with the hope of achieving a correlation of the results of ultrastructure research, the available information on the bioelectric activity, biochemical, and physico-chemical properties of nerve cells." Among the AAN members invited to attend are: T. H. Bullock, J. Folch-Pi, Samuel T. Hicks, S. L. Palay, F. I. Schmitt, and Paul Weiss....The Venezuelan Institute is situated on a picturesque mountain top, 5,000 feet above Caracas but only seven miles away from the center of the city. Under the leadership of H. Fernandez-Moran, the institute has passed the "pilot" stage of its development and is forging ahead into what appears to be a promising future.

Roosevelt Hotel Center for N. Y. Neurological Meetings The traditional series of pre-Christmas neurologic meetings were held in New York (Dec. 6-11), most of them at the Hotel Roosevelt. The American League Against Epilepsy held a one-day meeting (Dec. 6), featuring innovations in seizure control; the prognostic value of the EEG; and insights into the future understanding of seizures gathered from clinical medicine (Walter O. Klingman) and from biochemistry (Harold E. Himwich). The sessions were opened by William G. Lennox, "A Century of Medical Therapy in Epilepsy," and the presidential address was given by James L. O'Leary....Also at the Roosevelt (Dec. 7-8), the Association for Research in Nervous and Mental Disease held its thirty-sixth annual meeting. The subject under consideration was the brain and human behavior. Present as a distinguished guest speaker was Frederic Bremer, professor of pathology at the University of Brussels, president of the Fourth International Congress of Electroencephalography and Clinical Neurophysiology, and honorary-corresponding member of the AAN. Prof. Bremer presented a paper on the physiology of the corpus callosum....AAN members serving on the A.R.N.M.D. Commission at this meeting were: M. B. Bender, Harold D. Himwich, Seymour S. Kety, Heinrich Kluver, Horace W. Magoun, and David McK. Rioch.... Other related meetings held in New York were the Medical Advisory Board (Kermit E. Osserman, secretary) of the Myasthenia Gravis Foundation at the Roosevelt (December 7), which included scientific discussions on recent advances in myasthenia gravis; the Medical Advisory Board (Augustus S. Rose, chairman) of the National Multiple Sclerosis Society, at the Hotel Biltmore, December 9; and the annual examinations of the American Board of Psychiatry and Neurology, which took place at the Columbia-Presbyterian Medical Center (December 10-11).

Cerebral Palsy Group Meets in Chicago The tenth annual session of the American Academy for Cerebral Palsy was held in the Congress Hotel, Chicago (November 17-19, 1956). Among the highlights of the meeting was a symposium on basic research under the chairmanship of Glidden L. Brooks. Participating were: Angel Pentschew (pathology), Leonard T. Kurland (epidemiology), and William F. Windle (asphyxia neonatorum)....The new officers of the AACP are: Nicholson J. Eastman, president; William T. Green, president-elect; and Raymond R. Rembolt, secretary-treasurer....The 1957 meeting will be held at the Roosevelt Hotel in New Orleans (November 25-27). The day preceding the meeting will be devoted to a symposium on kernicterus, organized by the society's research committee (Chester Swinyard, chairman).

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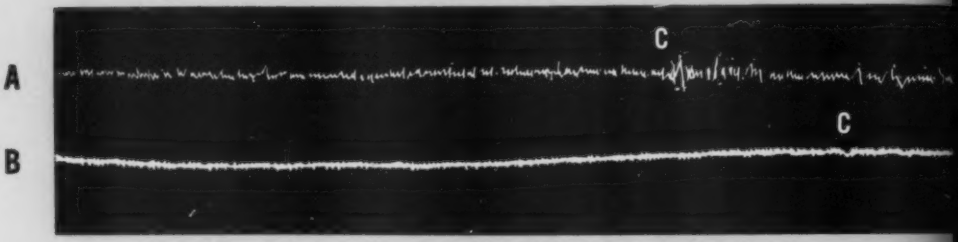

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1. Dickel, H.A., et al.: West. J. Surg., April, 1956.

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